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THE SURGICAL RELIEF OF MITRAL STENOSIS: A PRELIMINARY REPORT.

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CHRONIC rheumatic valvular disease is generally thought to affect about 0·5% to 1·0% of any community where acute rheumatic fever is highly endemic (White, 1946). Of 100,000 Americans rejected for service in the second World War because of cardio-vascular disorders, at least half suffered from rheumatic valvular defects (Levy, Stroud and White, 1943). Uncomplicated mitral stenosis accounts for about 50% of all rheumatic valvular lesions, so that in Sydney, where the incidence of chronic rheumatic carditis is about 0·6%, there are about 4000 people with mitral stenosis. The chief factor in the production of symptoms in mitral stenosis is the mechanical block to the intracardiac circulation, resulting from the fusion of the leaflets of the mitral valve. Other anatomical abnormalities, such as shortening of the *chordæ tendineæ*, calcification of the valve leaflets, and fibrosis of the valve ring and of the myocardium probably play a subsidiary role in the majority of cases. It was natural that such a simple mechanical fault should attract the attention of surgeons, and as early as 1902 Sir Lauder Brunton, a physician, advocated separation of the cusps along their line of pathological fusion. In 1926 Souttar treated one patient by this method.

The first surgical attempt to relieve the obstruction was made by Cutler in 1923. Subsequent experience with the early procedures has been reviewed by Bailey, Glover and O'Neill (1950). It was not generally appreciated until quite recently that the bad surgical results of the 1920's and 1930's were due not to the failure to relieve the obstruction (in fact this was done only partially), but to the production of mitral regurgitation. Murray (1950) overcame this problem by inserting a new valve fashioned from a vein, and Bailey (1949), and Baker, Brock, and Campbell (1950) achieved similar results by splitting the valve along the line of fusion (commissurotomy) precisely as Brunton had suggested. Early experience with these methods of attack has been satisfactory, and this paper reports the short-term results in the first six cases in which the Bailey-Brock procedure was adopted in the Royal Prince Alfred Hospital, Sydney.

The Present Series.

The clinical, radiological, electrocardiographic and operative findings are summarized in Tables I and II. All these patients had severe mitral stenosis with a very limited functional capacity and were quite unable to carry on their duties. The first patient had been given an invalid pension in 1944, and when first examined by us was unable to climb more than a few stairs at a time. He had been having severe hæmoptyses for seven years, and over the last year his physician, Dr. P. C. P. Waugh, had treated

him for many attacks of acute pulmonary œdema. The second patient was the most severely affected of the series, and became dyspnœic when she changed her clothes. She was the only one of the six who had been observed to have frank congestive failure. Of the remainder, the third, fourth and fifth patients were comparable with the first, and the last patient was somewhat better, although still greatly handicapped. In the fifth patient two pregnancies had been terminated because of heart disease.

Three of the patients had a regular pulse. The first patient had had paroxysmal attacks of auricular fibrillation, and the fourth and sixth patients had auricular fibrillation of some years' duration. All of them had been given digitalis by their physicians. The cardiac signs were quite characteristic of classical mitral stenosis. The mitral diastolic or presystolic murmur, the accentuated first sound at the apex and the accentuation of the pulmonary second sound, sometimes with splitting, were found in every patient. None had pulmonary or aortic diastolic murmurs. The right ventricle was hypertrophied as judged by palpation of the precordium in five of the six cases.

The radiological changes were also characteristic in showing dilatation of the left auricle and pulmonary artery in all. The right ventricle was also thought to be hypertrophied in every case, when observed in the left oblique view. All the patients had pulmonary venous congestion, and two were thought to have pulmonary hæmosiderosis (Figure I). Calcification of the mitral ring or cusps was not observed fluoroscopically in any patient.

Tall, notched or broadened *P* waves were recorded on the electrocardiogram of three of the four patients with sinus rhythm. The heart was vertical or semi-vertical in all, and the *QRS* complex showed evidence of right ventricular hypertrophy in the unipolar chest leads in four. One showed incomplete right bundle branch block (Figure II), and in the last patient the precordial leads were normal.

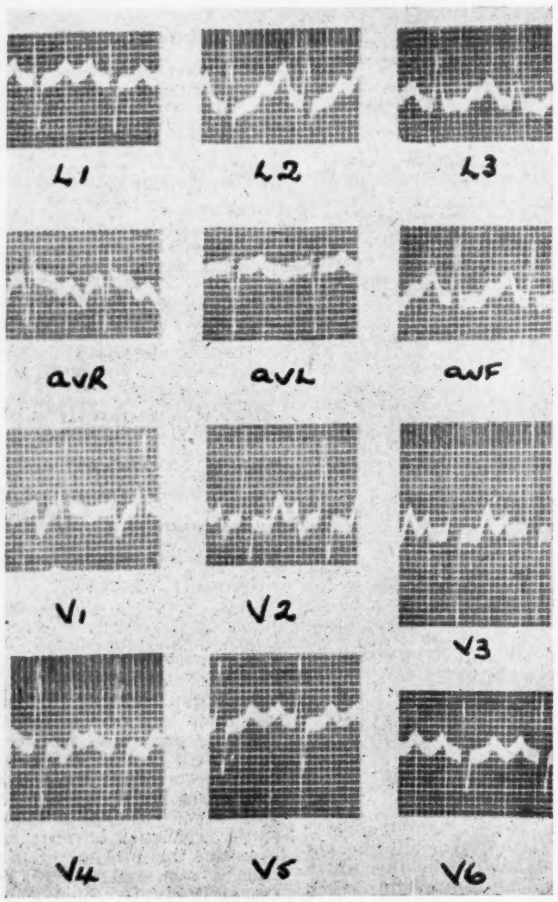

FIGURE II.
Electrocardiogram from Case II, showing incomplete right bundle branch block.

Functional Studies.

When one examines numbers of patients with pure mitral stenosis, the difficulties of precise functional assessment are often very real. The history and the exercise tolerance, the presence or absence of pulmonary congestion, the size of the pulmonary artery, the intensity of the pulmonary second sound and evidence of right ventricular hypertrophy must all be taken into account; but even then judgement is often difficult. A simple exercise tolerance test affords much valuable information, but leaves many questions unanswered. We have therefore adopted the policy of directly measuring the pulmonary artery and "capillary" pressure (Hellems, Haynes and Dexter, 1949) and cardiac output at rest and during exercise by the technique of cardiac catheterization (Cournand and Ranges, 1942). Although it is subject to the same limitations as any other functional test requiring the cooperation of the unanæsthetized patient, most of the pitfalls can be recognized with experience and allowance made. If one has the complete confidence of the patient, the errors of the method are relatively small and the results are reproducible. The pre-operative data from four of the six patients treated surgically are summarized in Table III.

At rest all these patients had a raised arterio-venous oxygen difference with a normal arterial oxygen saturation. The oxygen consumption was normal and the cardiac outputs and indices were about two-thirds of normal. The pulmonary capillary, pulmonary artery and right ventricular systolic pressures were all raised. The right ventricular end diastolic and right auricular mean pressures were normal in three of the cases and doubtfully raised in Case II. On mild exercise, which varied with the capabilities of the individual patient, but which did not exceed three minutes' duration, the pulmonary arterial pressure rose rapidly to a much higher level and returned at different rates to the resting values. In the two cases in which simultaneous studies of the cardiac output were carried out the already abnormal venous unsaturation increased even more and the oxygen consumption rose less than normal, so that the cardiac output remained essentially unchanged. In such patients the increased venous return of muscular exercise results only in an increased accumulation of blood in the lungs, severe dyspnœa, to which it is doubtless related, muscular fatigue and, on occasions, pulmonary œdema. Similar changes are found in pulmonary hypertension secondary to left ventricular failure, but this can usually be ruled out on clinical grounds. Resting pressures without outputs and studies of the response to exercise are apt to be misleading, for we have found equally high resting pressures in patients with normal outputs and a good response to exercise.

Our findings agree broadly with those of Bayliss, Etheridge and Hyman (1950) and of Gorlin and others (1951*a*, 1951*b*). In most reported cases of severe mitral stenosis the resting cardiac output is about half to two-thirds of normal, and the extraction of oxygen from the blood by the tissues is raised above normal. Exercise leads to an increase in the oxygen extraction and the oxygen consumption, with no significant change in the

TABLE I.

The Symptoms and Physical Signs in Six Cases of Mitral Stenosis.

Case Number.	Age. (Years.)	Sex.	Duration of Condition. (Years.)	Symptoms.				Physical Signs.							
				Hæmoptysis	Acute Pulmonary Œdema.	History of Congestive Cardiac Failure.	Classification. [1]	Pulse Rate per Minute.	Rhythm.	Blood Pressure. (Millimetres of Mercury Systolic/Diastolic.)	Apex Beat.	Right Ventricular Hypertrophy.	Apical Thrill.	Mitral Area.			Pulmonary Second Sound.
														First Sound.	Systolic Murmur.	Diastolic Murmur.	
I	36	M.	9	Severe recurrent.	Many attacks.	Absent.	III	80	Regular.	140/85	Fifth left intercostal space, four and a half inches from the mid-line.	Absent.	Absent.	Accentuated.	Absent.	Present.	Strongly accentuated.
II	27	F.	8	Streaking only.	+++	Yes.	IV	110	Regular.	125/80	Fifth left intercostal space, outside mid-clavicular line.	++	Absent.	Markedly accentuated.	Grade II	Present.	Strongly accentuated. Widely split.
III	27	F.	5	Severe.	+++	Absent.	III	90	Regular.	105/70	Fifth left intercostal space, four and a half inches from the mid-line.	+	Presystolic.	Accentuated.	Absent.	Present.	Accentuated. Split.
IV	31	F.	12	None.	Six attacks.	Yes.	III	96	Irregular.	125/80	Fifth left intercostal space, four and a half inches from the mid-line.	+	Diastolic.	Accentuated.	Grade I.	Loud.	Accentuated. Split.
IV	32	F.	8	Severe.	No definite attack.	No definite attack.	III	110	Regular.	120/70	Fifth left intercostal space, four and a half inches from the mid-line.	+	Diastolic.	Accentuated.	Grade II.	Present.	Strongly accentuated. Split.
VI	37	F.	12	None.	None.	Absent.	III	80	Irregular.	130/90	Fourth left intercostal space, between mid-clavicular and anterior axillary lines.	+	Absent.	Accentuated.	Grade I.	Present.	Accentuated.

Systolic murmurs are graded from I to V, Grade I being the mildest. [1] The functional classification used is that of the New York Heart Association.

TABLE II.

Radiological, Electrocardiographic, Operative and Post-Operative Findings in Six Cases of Mitral Stenosis.

Case Number.	Radiological Signs.					Electrocardiogram.				Operative Findings.		Post-Operative Comments.			
	Cardiac Enlargement	Right Auricle.	Right Ventricle.	Left Ventricle.	Left Auricle.	Pulmonary Artery.	Lung Fields.	Rhythm.	Electrical Position.	*P* Waves.	*QRS* Complex.	Approximate Diameter of Mitral Orifice.	Extent of Splitting of Commissures.	Time Since Operation.	Functional Result.	Signs of Mitral Stenosis
I	++	N.	++	N.	+	+	Congested, haemosiderosis.	Sinus.	Vertical.	Notched and broadened.	Right ventricular hypertrophy.	8 millimetres.	Anterior complete, posterior partial.	5 months.	Excellent.	Present.
II	+	+	++	N.	++	+	Congested, haemosiderosis.	Sinus.	Vertical.	Notched, broadened, 4 millimetres high.	Incomplete right bundle branch block.	4 millimetres.	Anterior complete, posterior not at all.	4 months.	Fair.	Present.
III	++	N.	+	N.	+	++	Congested.	Sinus.	Vertical.	++	Right ventricular strain.	10 millimetres.	Anterior complete, posterior partial.	3 months.	Good.	Present.
IV	++	+	++	N.	++	+++	Congested.	Auricular fibrillation.	Vertical.	Absent.	Right ventricular hypertrophy. Digitalis effect.	6 to 8 millimetres.	Anterior complete, posterior partial.	2 months.	Good.	Present.
V	+	N.	+	N.	++	++	Congested.	Sinus.	Vertical.	Notched, broadened.	Right ventricular hypertrophy and strain.	6 to 8 millimetres.	Anterior complete, posterior partial.	2 months.	Good.	Present. Also slight regurgitation.
VI	++	N.	+	? +	++	++	Congested.	Auricular fibrillation.	Semivertical.	Absent.	Within normal limits.	10 to 12 millimetres.	Both commissures complete.	1 month.	Good.	Present.

TABLE III.

Some Measurements of the Circulation in Four Patients with Mitral Stenosis at Rest.

Case Number, Sex and Age (Years).	Height (Inches) and Weight (Pounds).	Body Surface Area. (Square Metres.)	Oxygen Content, Mixed Venous Blood (Volumes *per Centum*). (*a*) Rest, (*b*) Exercise.	Oxygen Content, Arterial Blood (Volumes *per Centum*). (*a*) Rest, (*b*) Exercise.	Oxygen Saturation of Arterial Blood (*per Centum*). (*a*) Rest, (*b*) Exercise.	Arterio-Venous Oxygen Difference in Volumes (*per Centum*). (*a*) Rest, (*b*) Exercise.	Oxygen Consumption. (Millilitres per Minute.) (*a*) Rest, (*b*) Exercise.	Cardiac Output. (Litres per Minute.) (*a*) Rest, (*b*) Exercise.	Cardiac Index.[1] (*a*) Rest, (*b*) Exercise.	Pressures in Millimetres of Mercury.			
										Pulmonary Capillary Mean. (*a*) Rest, (*b*) Exercise.	Pulmonary Artery. (*a*) Rest, (*b*) Exercise.	Right Ventricle. (*a*) Rest, (*b*) Exercise.	Right Auricle. (*a*) Rest, (*b*) Exercise.
I; M., 36	75, 175	2·06	(*a*) 10·4 (*b*) —	(*a*) 17·4 (*b*) —	(*a*) 96 (*b*) —	(*a*) 7·0 (*b*) —	(*a*) 288 (*b*) —	(*a*) 4·1 (*b*) —	(*a*) 2·0 (*b*) —	(*a*) 15 (*b*) —	(*a*) 60/15, Mean 33. (*b*) 100/36 Mean 63	(*a*) 60/1 (*b*) —	(*a*) 0 (*b*) —
II, F., 27	59½, 100	1·37	(*a*) 9·7	(*a*) 17·1	(*a*) 94	(*a*) 7·4	(*a*) 189	(*a*) 2·7	(*a*) 1·9	(*a*) 18	(*a*) 70/48 Mean 57	(*a*) 70/8 Mean 42	(*a*) 6
V, F., 32	67½, 133	1·70	(*a*) 10·5 (*b*) 7·5	(*a*) 16·5 (*b*) 16·5	(*a*) 96 (*b*) 96	(*a*) 6·0 (*b*) 9·0	(*a*) 215 (*b*) 323	(*a*) 3·6 (*b*) 3·2	(*a*) 2·1 (*b*) 2·1	(*a*) 41 (*b*) —	(*a*) 78/46 Mean 60 (*b*) 134/76 Mean 104	(*a*) 78/5 Mean 30 (*b*) —	(*a*) 5 (*b*) —
VI, F., 37	63½, 155	1·73	(*a*) 12·5 (*b*) 10·0	(*a*) 18·4 (*b*) 18·6	(*a*) 93 (*b*) 94	(*a*) 5·9 (*b*) 8·6	(*a*) 226 (*b*) 379	(*a*) 3·8 (*b*) 4·4	(*a*) 2·2 (*b*) 2·6	(*a*) 21 (*b*) —	(*a*) 40/26 Mean 30 (*b*) 54/50 Mean 44	(*a*) 36/4 Mean 6 (*b*) —	(*a*) 2 (*b*) 2

[1] The cardiac index is the cardiac output in litres per square metre of body surface per minute. Reference point for pressures was 5·0 centimetres behind the sternal angle.

cardiac output. It must also lead to an immediate increase in venous return, and provided the right ventricle is competent, blood must accumulate in the pulmonary vasculature, where the pressure rises. The rises in pulmonary arterial and capillary pressure are approximately the same (Gorlin *et alii*, 1951*b*), and as the latter pressure agrees to within a few millimetres of mercury with the left atrial pressure (Dow and Gorlin, 1950), it can be reasonably assumed that the left atrial pressure rises by a similar amount. If the left ventricle is competent an increase in the filling pressure should result in an increase in cardiac output; in mild mitral stenosis this usually occurs, but in severe disease the tachycardia of exercise so shortens the diastolic filling period of the left ventricle that the effective filling alters very little. Blood is therefore trapped in the lungs, the pressure rises, and if the exercise or the stenosis is severe enough, the resultant increase in pulmonary capillary filtration will result in pulmonary œdema. Many of the symptoms and signs of mitral stenosis are due to pulmonary hypertension and its further elevation on exercise, and direct measurement of the pressure provides the best objective evidence we have of the severity of the lesion. Some patients die of pulmonary œdema or infection, which increases pulmonary hypertension, while in others failure and dilatation of the right ventricle as a result of many years of excess work against pressure lead to the familiar picture of congestive cardiac failure, complicated often by tricuspid incompetence.

Selection of Patients.

We have been guided in the selection of patients for operation mainly by the experience of Glover, O'Neill and Bailey (1950) and of Baker, Brock and Campbell (1950). It often happens that when a new surgical procedure is being developed the surgeon is presented with patients so near to death that there is little hope of success; the miraculous recovery of a small proportion of his patients then leads to wider recognition of the therapeutic possibilities and the opportunity to operate in more favourable cases. This has certainly been true of mitral stenosis, and it is largely owing to the experience gained by Bailey, Brock, Harken and their colleagues that we have been able to avoid the usual early disappointments. On the other hand, in recommending a new procedure, the risks of which in our hands were not known but were conceivably great, it did not seem wise to select patients who were able to go about their daily tasks with only slight inconvenience. In steering between the Scylla of hopeless cases and the Charybdis of unnecessary operation we have perhaps chosen patients not quite so ill as some of those operated on by Brock. Nevertheless the cases were all severe but uncomplicated, and it remains to be seen whether the long-term results are good enough to warrant the policy of operating only in severe mitral stenosis. At this early stage the results certainly appear favourable, though certain surgical difficulties to be mentioned later suggest that there may be arguments for earlier operation.

There are some contraindications to operation, and these will be discussed first.

Acute or subacute rheumatic activity may be difficult to detect; but if it is suspected, operation should not be carried out. Nearly all the patients so far operated on abroad and all those treated here have been aged over twenty years; most of them have been nearer thirty years when recrudescence of rheumatic fever is less likely. It is clearly unwise to split the commissures if the process which resulted in their fusion is still active or likely to recur; on the same grounds subacute bacterial endocarditis is a contraindication. Nevertheless there will be young patients with severe mitral stenosis in whom the risk of recrudescence of rheumatic fever will have to be neglected and operation carried out; the dangers of acute rheumatic carditis are clearly greater if severe mitral stenosis is unrelieved.

Combined aortic and mitral valvular lesions occur nearly as frequently as mitral stenosis alone, and patients with double lesions form a good percentage of those referred to us. Gross aortic valvular lesions are, of course, a contraindication, but it is not always easy to decide whether an aortic diastolic murmur is significant from the point of view of operation. Although none of the present series of patients had any detectable aortic stenosis or incompetence, there is no obvious reason why mild aortic lesions should be a disqualification. Indeed, restoration of the function of the anterior leaflets by commissurotomy may lead to better left ventricular ejection and to a reduction of aortic regurgitation during diastole. On the other hand, a low cardiac output secondary to severe mitral stenosis may lessen the apparent severity of aortic stenosis. Relief of the stenosis with an increase in left ventricular filling and cardiac output will lead to an accentuation of the signs of aortic stenosis. This lesion alone has, on the whole, a good prognosis, and it is reasonable to expect that if accompanying mitral stenosis is relieved, worthwhile benefit may follow.

Tricuspid stenosis, when it occurs, is usually accompanied by mitral stenosis (Pitt, 1909) and often by aortic valve disease as well. When it is the dominant lesion pulmonary congestion is absent in spite of obvious dis-

ability, and at catheterization right ventricular and pulmonary artery pressures are normal at rest and during exercise. Characteristic auricular waves are seen in the jugular venous pulse (Mackenzie, 1913).

The diagnosis of mitral regurgitation has had many vicissitudes and has in some quarters been out of favour. Though usually found in association with stenosis, it has features of its own which have not yet been adequately explored; it is probably responsible for most cases of aneurysmal dilatation of the left auricle (Wood, 1951). The clinical diagnosis of the less advanced lesions has never been easy, and most clinicians have relied on the loudness of the mitral systolic murmur and evidence of left ventricular hypertrophy. Latterly pulsation of the left auricle during ventricular systole has been given attention. Mitral systolic murmurs may be misleading, and we have found patients with undoubted regurgitation and little or no systolic murmur and *vice versa*. In the presence of mitral stenosis producing right ventricular hypertrophy, we have not always found it easy to say whether or not the left ventricle is enlarged; finally pulsation of the left auricle has yet to be properly evaluated. Pathologically the lesion is doubtless associated with shortening of the *chordæ tendineæ*, and commissurotomy could not remedy this. In addition, operations aimed at relieving stenosis by producing regurgitation have been highly lethal. Therefore surgeons abroad now refuse operation when regurgitation is severe. As most patients with stenosis have some regurgitation, it is often a nice point to decide whether or not the regurgitation is sufficient to contraindicate surgery; in many cases it appears that regurgitation in mitral stenosis is lessened by valvulotomy, as the valve works better. There is at present no objective measurement of the degree of regurgitation, but its presence can be shown and roughly estimated on the pulmonary "capillary" pressure tracing (Lagerlof and Werko, 1949).

In long-standing mitral stenosis obliterative arteritis of the pulmonary vasculature (Parker and Weiss, 1936) adds to the burden on the right ventricle. Pulmonary hypertension then depends on obstruction to flow at the mitral orifice together with obstruction due to pulmonary arteriolar narrowing. If the latter factor becomes sufficiently important, then the results of mitral valvulotomy will depend on the reversibility of the pulmonary arteriolar changes. Whether or not they are entirely anatomical is not clear; in practice these patients do not do well (Murray, personal communication), and some surgeons therefore refuse operation if the pulmonary systolic pressure at rest is over 100 millimetres of mercury. Such cases are in the minority, and it is perhaps too early to adopt such a rigorous standard.

Congestive cardiac failure, seen so commonly among mitral stenotics in the medical wards of a teaching hospital, is at present a contraindication unless it can be relieved. Already three patients have died in this state while awaiting operation. Our second patient developed a small pulmonary infarct following catheterization and had an episode of failure. She subsequently stood operation very well. Provided that the surgical risk can be overcome there seems to be no reason why an incompetent right ventricle, on which systemic congestion depends, should not recover at least partially after commissurotomy.

If the patient with the very large heart, with established right-sided failure, with free mitral regurgitation, with significant aortic valve disease or with active rheumatic carditis is refused operation, the results should be good. The ideal patient for surgery is one who is aged between thirty and forty-five years, who is not in a state of congestive failure and who has no history of severe congestion, but who has had recurrent "bronchitis", pulmonary œdema or hæmoptysis and is seriously incapacitated (Class III of the New York Heart Association Classification, 1946). Such a patient has classical signs of mitral stenosis and objective evidence of pulmonary hypertension and right ventricular hypertrophy. If only such patients are chosen the operative mortality should be negligible. Auricular fibrillation is not necessarily a contraindication, but it makes the operative risk greater because of possible thrombosis in the left auricle, and the degree of functional recovery is likely to be less, though still worth while. Hæmoptysis alone is apt to be misleading. We have patients who had hæmoptysis many years ago which was undoubtedly due to mitral stenosis; their functional capacity now is still too good to warrant operation.

Surgical Aspects.

The surgical pathology and technique of operation have been discussed in detail in the publications of Baker, Brock and Campbell (1950) and of Bailey, Glover and O'Neill (1950). The rheumatic process leads to a thickening and adhesion of the two flaps of the mitral valve just proximal to their free edge. The thickening is usually irregular and tends to be greatest at the edges of the stenosed orifice. The adhesion leads classically to a button-hole orifice which in its extreme degree measures only a few millimetres in diameter. The orifice often assumes different shapes, but its axis corresponds to that of the normal valve. Although the mitral ring itself may contract, the adjacent parts of the cusps are often less diseased than the parts that have adhered and even calcified. Splitting of the valve along the line of pathological fusion will therefore lead to considerable restoration of function. Nearly all surgeons experienced in mitral valve surgery now agree that this is the method of choice. It has been found that mitral regurgitation, if not present before operation, is usually absent after it. Splitting may be done with the finger in many cases, but if this does not occur readily, then a knife must be used. It is particularly important to avoid cutting the cusps, as mitral regurgitation is then inevitable. This is particularly true of the antero-medial cusp, and division of this structure in dogs leads to a high mortality (Harken *et alii*, 1950).

Closed-circuit anæsthesia with controlled respiration is employed as a routine measure, "Pentothal" and curare being used with cyclopropane or nitrous oxide and ether. Dr. W. I. T. Hotten, who is responsible for the anæsthesia, states that there are fewer problems than in cases of lung suppuration or carcinoma with retained secretions, and we have had no anæsthetic difficulties. We have used procaine solution (0·4%) given intravenously at the rate of one gramme per hour to diminish cardiac irritability, and have also injected five millilitres of 4% procaine solution into the pericardial cavity before opening it five minutes later.

The surgical approach is either by an anterior incision in the third left intercostal space with division of the third and fourth costal cartilages at their junction with the sternum, or by a postero-lateral approach through the bed of the left fifth rib, the patient lying on the right side. This latter method has been used exclusively in the cases reported here.

After the lung has been retracted the mitral valve is approached from the left atrium. By far the best access is provided by the left auricular appendage. During the five minutes following the instillation of procaine solution into the pericardial sac the heart and great vessels are inspected. The pulmonary artery is always found to be dilated and under increased pressure, and the lungs are firmer than usual. The pericardium is incised vertically behind the phrenic nerve, and the auricular appendage is examined and palpated for clots. A special non-crushing clamp is placed across the base of the appendage and a pursestring suture inserted just above it. The auricular appendage is now opened towards its apex by an incision large enough to admit the index finger. The finger is inserted, the assistant removes the clamp and the finger is pushed into the atrium while another assistant tightens the pursestring to make a seal round the finger. The orifice is usually very small and sometimes not easily found. The thickness and consistency of the valve cusps and the size of the orifice are now estimated, and the direction of the commissures is noted. They are almost vertical with the patient in the lateral position. If mitral regurgitation is present the returning jet of blood can be felt during ventricular systole. An attempt is now made to separate the valve cusps at the commissures with the finger. If this is not possible, the finger should be withdrawn and a small commissurotomy knife fashioned to fit the curve of

the finger inserted. With the knife it is usually easy to slit the valve and then carry out digital dilatation right out to the muscular wall of the heart. During these manœuvres special care must be taken not to obstruct the mitral orifice for more than two or three beats, otherwise the blood pressure falls to unreadable levels. In addition, arrhythmias, usually simple extrasystoles, but sometimes short runs of ventricular tachycardia, may be provoked by the finger in the ventricle. When the surgeon is satisfied that no more can be done, the degree of mitral regurgitation is estimated by letting the finger lie in the atrium for a few beats. The finger is now removed and the clamp is applied. The auricular appendage is amputated, the cut margin is oversewn and the pursestring suture is tied. The pericardium is now completely closed and the chest is closed with drainage.

We have been particularly careful to replace blood promptly when it is lost and have had a slow intravenous drip transfusion begun with the anæsthetic. It is not always easy to decide how much blood is lost when it spurts for a few seconds from the heart and is quickly removed by assistants. The danger is to over-estimate the loss and give too much; these patients are close to pulmonary œdema, and overloading of the circulation probably carries greater risks than the loss of, say, one pint. With experience the loss of blood is negligible. We have also been apprehensive of arrhythmias developing during surgery; although some arrhythmia develops in almost every case, it is usually of no great consequence. In our cases an electrocardiograph was connected to the patient before the operation began, and it has given some interesting but probably not essential information to the surgeon. The most anxious time is during the actual dilatation, when ventricular tachycardia, complete heart block, nodal rhythm and transient auricular fibrillation have all been recorded.

The patient is usually semi-conscious by the end of the operation and is moved to the ward, where an oxygen tent is waiting. He is nursed in the upright position in bed, and so far there have been no major post-operative worries. The first patient developed paroxysms of auricular fibrillation within forty-eight hours of operation, and this rhythm subsequently became established. Two patients operated on since this series developed auricular fibrillation which reverted to normal rhythm. It was found that digitalis had been omitted in both the latter, and when it was given again the rhythm reverted within forty-eight hours. It would seem that however successful the valve slitting, digitalis administration should be continued into convalescence. We have used quinidine only for post-operative fibrillation in the first case; the effective dose was too high to be tolerated.

Results of Operation.

The immediate short-term results have been most promising, although no patient of this series can be said to be cured of mitral stenosis. All the patients still have the characteristic murmurs. The first patient, operated on in March, 1951, has shown a remarkable change. He had been an invalid pensioner for nearly seven years when first examined by us, and was unable to walk more than a hundred yards on the flat. He had had repeated attacks of acute pulmonary œdema and severe hæmoptysis over the last year, and was prepared to accept almost any risk to get well. Since operation he has lost all his symptoms, has had no further hæmoptysis, can run 100 yards without undue trouble and has returned to work. The remainder of the patients tell similar stories. Their hands are warmer, their pulses are stronger and we infer that their cardiac output is now normal.

We have not catheterized the hearts of any of these patients since operation. There are very few published cases studied post-operatively, and although these have shown in general a fall in pulmonary vascular pressure and resistance, the changes in other respects are too equivocal to be convincing.

Discussion.

It is clear from our short experience that this operation can produce remarkable improvement in some patients with severe mitral stenosis. The mortality in selected cases is negligible and the convalescence short. There are certain surgical factors not mentioned so far or encountered in this series which lessen the prospects of success. The first is the possibility of finding an auricular appendage so small that it will not give sufficient access to the mitral valve. We know of no way of avoiding this or of warning the surgeon of its presence. The alternative pulmonary vein method of approach is not easy. In such cases perhaps another technique may be best—for example, that of Murray, the left ventricular approach being used.

The dangers of mitral regurgitation have been stressed, and although it should not be a problem in this operation, at least one of our patients, although certainly much improved, has more regurgitation following operation. Her commissurotomy was incomplete, and if it had been more effective regurgitation probably would not have occurred.

The well-known association of mitral stenosis with embolism and auricular fibrillation has recently been stressed by Mattingly *et alii* (1950); of 164 patients with mitral stenosis and embolism, 140 had auricular fibrillation. Seventy-one died as the result of embolism, half at the initial episode. In only eight of 36 autopsies was the thrombus confined entirely to the left auricular appendage. These figures are in general agreement with the earlier ones of Harris and Levine (1941). The average age in the latter series was forty-three years, and 55 of the 72 patients had auricular fibrillation. Although embolism is not uncommon among patients examined in hospital, it is clear from the foregoing figures that it usually occurs late in the course of mitral stenosis. The embolus is almost invariably derived from a thrombus taking origin in the left auricular appendage. Thrombosis has for long been related by pathologists to stasis and endothelial damage. The part played by the latter factor in the auricular appendage is probably small, and it would seem that stasis is the main determinant of thrombosis. We know from catheter studies that mitral stenosis may exist for years without any fall in the cardiac output at rest and still with an appreciable rise on exercise. The probability is that such people do not have thrombosis or embolism. The clinical evidence is that these complications occur in patients with severe mitral stenosis, most often in the presence of auricular fibrillation, with a poor exercise tolerance and probably with a low cardiac output. It is therefore an indication that something must be done for the mitral stenosis. Unfortunately we know of no way of determining the extent of the thrombus or even whether one is present or not in the absence of embolism. Auricular fibrillation and a large left auricle make its presence highly likely. One must therefore conclude that, provided there are no other contraindications, the rational procedure is to explore the left auricle in the hope that the thrombus is small and that valvulotomy is possible. Three of our patients did have a small clot in the left auricle. If a large clot is found in the auricle, the operation is perhaps best abandoned. Attempts to find a passage through adherent and partially organized clot or to dissect the clot away from the auricular wall will probably end disastrously by causing embolism or the formation of a "ball valve" clot which may cause sudden death. Amputation of the auricular appendage alone is not recommended, as in the majority of cases the clot has extended further.

Although this operation is now performed in many parts of the world, it has been developed so recently that no long-term follow-up is yet possible. In Bailey's first two successful cases the operation was performed about three years ago, and as far as is known their improvement has been maintained. Brock's first patient was treated a few months later, and she too has continued to do well. But there are likely to be disappointments. The cardiac muscle may not recover sufficiently in some, and the possibility that the cusps will again adhere has also been suggested with some evidence to support it (Magarey, 1951). Basically the concept of relieving the obstruction to the circulation is a sound one, and unlike the treatment offered for some other medical diseases, this treatment is aimed directly at the relief of the abnormality. It is now quite certain that pulmonary congestion and œdema,

dyspnœa, repeated hæmoptysis and ultimately right-sided heart failure depend directly in the majority of cases on the increased resistance to flow at the mitral valve, and once these signs and symptoms appear in the natural course of rheumatic carditis, medical treatment has little to offer but many years of chronic invalidism. Relief of symptoms and restoration to normal or limited activity, if only for a few years, are eminently worth while. Now that relief is possible, every effort should be made to see that patients with mitral stenosis are not allowed to drift into the inoperable stage without consideration of surgical treatment. Even then there is the prospect that the more general application of the principle and practice of Murray may save patients considered unsuitable for the procedure we have used. Murray's patients have all been far more advanced in their disease process than any we have treated, and the left ventricular approach may prove in the long run to be the most suitable for patients with large left auricles, much clot formation and a considerable degree of mitral incompetence.

Summary.

The clinical and functional findings in the first six patients operated on in this hospital for mitral stenosis are presented. The first patient was treated in March, 1951. There have been no deaths, and the functional result has been very satisfactory in all.

Prior to operation all these patients were severely handicapped and unable to carry out more than very slight exertion for longer than a minute or two. They all had pure mitral stenosis complicated in two cases by auricular fibrillation. They were chosen because they had no other significant valvular disease, evidence of mitral regurgitation, rheumatic activity or unduly large left auricles.

The severity of the lesion was demonstrated preoperatively by cardiac catheterization in four of the cases; we now carry out this procedure as a routine procedure before considering surgical treatment. The surgical technique has been described and the possible difficulties have been discussed.

Our own results and those of others abroad suggest strongly that every patient with mitral stenosis should be assessed on the assumption that sooner or later in the course of the disease surgical treatment may be of great benefit.

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Addendum.

Since this paper was written 17 more patients have been submitted to surgery for mitral valvulotomy, making a total of 23 in all. There have been two deaths. One patient with severe stenosis died during the induction of anæsthesia, although adequately prepared for surgery. The second death was in a patient with auricular fibrillation of several years' standing who died of embolism in the cerebral and iliac vessels on the third post-operative day. Two patients are not yet well enough for their improvement to be assessed. Of the remaining 19, 14 have shown great improvement, two are only slightly improved, and three, in whom for technical reasons dilatation was unsuccessful, are unimproved. The only noteworthy post-operative complication has been auricular fibrillation, in the control of which digitalis and particularly quinidine have been most effective.

TOXOPLASMOSIS: REVIEW OF THE LITERATURE AND REPORT OF A CASE.

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As only two cases of toxoplasmosis have been previously reported in this journal (Robertson, 1946), I have taken the opportunity in reporting this case to review the literature, with particular emphasis on the more recent serological diagnostic methods.

Toxoplasma gondii is a protozoan parasite first described by Nicolle and Manceaux in the North African rodent in 1908, and reported independently in the rabbit by Splendore from Brazil in the same year. "Toxin" is derived from the Greek and means "bow" or "arc", and refers to the lunate shape of the organism. The specific name "*gondii*" refers to the type of host—*Ctenodactylus gondii*.

Janku, in 1923, was the first to report human infection with the parasite, although he called the organism an encephalitozoon. In 1937 Wolf and Cowen described a human case, complete with clinical and pathological studies.

Epidemiology.

Cases of toxoplasmosis have now been reported from a number of countries, and from this it can be gathered that the disease is world-wide in incidence.

Infection in Animals.

Numerous animals, birds and reptiles, including the dog, cat, rabbit, sheep, guinea-pig, baboon, chimpanzee, rat, mouse, mole, canary, sparrow, domestic fowl, lizard and snake to mention a few have been stated to have exhibited spontaneous infection with toxoplasma. It may be mentioned that some writers have thrown doubt on the records in the literature of animal infectivity. Some of the statements made have been based on morphological grounds and

not on reproduction of the disease by transfer from the animal in question to an experimental animal. There are other protozoan parasites in birds and animals, such as the hæmogregarines, which morphologically simulate toxoplasma. Toxoplasma has also been confused with other protozoan parasites, particularly encephaliotzoon, leishmania and avian malaria.

Transmission.

As regards transmission from animal to animal and from animal to man there is less certainty. Ticks are regarded as possible vectors. There is good evidence that toxoplasma can come out in the urine and fæces of dogs and cats and so contaminate a person's hand and gain entry through the mouth. Transplacental infection explains congenital cases of the disease.

Morphology.

Morphologically toxoplasma is a sharply outlined crescentic organism measuring 4μ to 7μ in length and 2μ to 4μ in width. The cytoplasm is clear and distinct and a pronounced nuclear chromatin is present. The organism is non-motile and multiplies by binary fission. In addition to a single parasite, large aggregations or clusters of closely approximated organisms are observed in the tissues. Occasionally such collections appear to be surrounded by a cyst wall, and hence are referred to as cysts or pseudocysts. In this form it appears to cause no tissue reaction so long as the enveloping cell membrane is unbroken. Toxoplasma is an obligate, intracellular parasite and cannot be cultured on artificial media.

Clinical Forms of Toxoplasmosis.

Clinical forms of toxoplasmosis are as follows: (i) congenital or neonatal meningo-encephalitis associated with hydrocephaly, chorio-retinitis, cerebral calcification and convulsions; (ii) atypical encephalitis in older children; (iii) acute exanthematic infection in adults associated with pneumonitis and rash. Subclinical forms are recognized by the finding of specific antibodies in the blood of a mother who has given birth to an infected infant.

Clinical Manifestations of Infantile Toxoplasmosis.

The clinical signs of toxoplasmosis in children are variable. The widespread destruction of the central nervous system gives rise to the first clinical indication of the disease. Of infants affected by the disease 80% have internal hydrocephalus, which is obstructive in nature and indicates blockage of the flow in the subarachnoid space. Muscular twitching is present in 71% of cases and is followed by spasticity and generalized convulsions. Neck stiffness and head retraction are observed in only one-third of cases. Signs of meningeal involvement appear only in those cases in which severe damage has been done to the underlying nervous system. Changes in the meninges are secondary to the underlying brain infection.

The ocular signs are bilateral, focal and multiple. The macular region is invariably involved, and peripheral lesions also occur. Early lesions consist of localized œdema, indefinite demarcation from the surrounding retina and necrosis. Chorioidal atrophy may be present to such a degree that the underlying sclera may be visualized. Foci of atrophy are present with extensive pigmentary deposits at the periphery of the lesions. Older lesions consist of sharply marginated foci of chorioido-retinal atrophy with diffuse irregular pigmentation. The vitreous is not involved. Other eye changes are microphthalmos with retinal detachment, corneal atrophy and optic atrophy in varying degrees. Papillœdema may or may not be present. Toxoplasmic chorioido-retinitis must be differentiated from the following conditions: (i) Pseudoglioma. This is usually due to old chorioido-retinitis. The condition is usually unilateral and the macula is not invariably involved. (ii) Intraocular tumours, such as retinoblastomata. These occur mainly in children. The fundal changes in this condition are yellowish-white nodules extending in polypoid fashion into the vitreous. Pigmentation of the fundus is not a common feature. These newgrowths are often bilateral. In the later stage they may produce raised tension, and X-ray examination may reveal calcification in the lesions. (iii) Traumatic injuries at birth. These may cause hæmorrhages and subsequent scarring. The injuries are not usually bilateral and not selective to the macula. (iv) Cerebro-macular degeneration, such as Tay-Sachs disease. This presents both eye and brain changes. The eye changes are revealed as a cherry-red spot at the macula in each eye with whitish surrounding area. There is usually some pigmentation of the fundus. (v) Developmental defects, such as congenital colobomata. These, if involving the posterior eye segment, may simulate ocular lesions of toxoplasmosis. However, there is usually no evidence of inflammatory action in the lesions. (vi) Tuberous sclerosis. This is rare in infants and children. Again, in this disease we have a combination of fundal and cerebral changes. In the fundus are found white, striated, glistening areas of glial tissue over the retina, in which are numerous, small, highly refractile, whitish nodules. (vii) Acute metastatic chorioretinitis. In this condition a history of convalescence from an acute exanthem may be obtained. The fundal lesions are rapid in onset, small and irregularly scattered throughout the fundus, and uveitis generally ensues. (viii) Granulomata. These tumours occurring in children must be differentiated from ocular changes of toxoplasmosis. Ocular syphilis in children is congenital, and the fundal changes are diffuse, irregular, small areas of chorioiditis occupying most of the fundus. (ix) Tuberculous chorioiditis. In this condition the ocular lesions occur as terminal stages of miliary tuberculosis. The fundal changes are small, dull, yellowish focal areas scattered irregularly in the chorioid. (v) Retrolenticular fibroplasia. In this condition there is a history of prematurity with a birth weight about two pounds. A greyish mass is seen behind the lens, usually in each eye. The fundus is not usually involved.

Other signs of toxoplasmosis in children are jaundice occurring in about one-third of the cases and hepatomegaly and splenomegaly, present in about half the cases.

Adult Toxoplasmosis.

Adult toxoplasmosis is not a common condition. The onset is sudden, with chills, fever and elevated temperature. A maculo-papular rash covers the whole of the body except the palms of the hands and the soles of the feet. Pulmonary involvement is the outstanding feature. Diffuse interstitial pneumonitis with focal necrosis is found in the lungs *post mortem*. Death usually results from interference with the respiratory mechanism. Numerous parasites have been found in the lungs. Changes in the central nervous system are so slight that in most cases lumbar puncture has not been performed. There is no definite evidence that ocular lesions occur in adults as a result of post-natal infection.

It may be noted that in children the brunt of the damage falls on the immature central nervous system, whereas in adults the lungs bear the brunt of the disease.

Chronic Toxoplasmosis.

Cases of chronic toxoplasmosis have been recorded in which there was clinical or laboratory evidence of toxoplasmosis without signs of widespread activity. Toxoplasma has not been isolated in any such cases of chronic disease. The crucial question whether there is persistence of toxoplasmosis, hence true chronicity, must remain only partially answered. There is evidence to favour the hypothesis of a chronic stage of toxoplasmosis in human beings. On several occasions structures resembling toxoplasmic pseudocysts have been found in autopsy material examined as a routine procedure.

The recurrence of active chorioido-retinitis can best be explained in patients showing scars of the subacute form by the hypothesis that toxoplasmosis has persisted through a latent stage. Cases have been reported in which toxoplasmic chorioido-retinitis lesions became active during three subsequent pregnancies. Chronic toxoplasmosis has been demonstrated in certain animals in Nature and experimentally.

Pathology.

Central Nervous System Changes in Infantile Toxoplasmosis.

The pathological findings in the central nervous system are as follows.

Leptomeninges.—Examination of the pia-arachnoid overlying destructive lesions shows congestion of the vessels with infiltration by a large number of lymphocytes, plasma cells, mononuclear cells, fat-laden macrophages and eosinophile cells. This change is especially noted in the small arterioles, venules and capillaries. If the reaction is more severe there is obliteration of the sulci and gyri. Parasites are frequently found in the intimal cells of the arterioles, venules and capillaries.

Cerebral Cortex.—Examination of the cortex shows varying small foci of cellular infiltration in the grey matter to large areas of necrosis with extensive destruction of the brain substance. There is complete loss of architecture with large clusters of nuclear debris and numerous fat-laden macrophages and eosinophile cells. Necrotic cysts are often found. Microglial nodules are of common occurrence and toxoplasma organisms are found in them. The adjacent brain substance becomes œdematous and is infiltrated with lymphocytes. There is loss of neural and neuroglial elements with swelling of astrocytes and oligodendroglia. Examination of blood vessels shows hyperplasia of the intima. In infantile toxoplasmosis large foci of cerebral calcification are present within the zones of necrosis. The calcium salts are deposited as coarse granules.

Ventricles.—Ventricular changes are perivascular infiltration with lymphocytes and plasma cells, and ulceration of ependymal lining. Ulceration is frequently found in the cerebral aqueduct, where glial nodules and ulcerative lesions may occlude the lumen and so account for the internal hydrocephalus observed in infants.

Ocular Lesions.—Focal swelling of all layers of the retina and œdema and inflammatory necrosis are observed on examination of the fundus. Necrosis is observed mainly in the inner layer. There is usually diffuse infiltration with mononuclear cells and eosinophile cells near margins of foci. Examination of the blood vessels shows perivascularitis and hyperplasia of endothelial cells. Inflammatory swelling is usually present in the internal limiting membrane and is associated with ingrowth of fibroblasts and capillaries into the adjacent vitreous. Many of the nerve cells are destroyed. The chorioid vessels and optic nerve are moderately infiltrated with plasma cells, lymphocytes and mononuclear cells. Parasites are usually found in large numbers within the retina, especially in the necrotic foci and chorioid.

Comment.—The constant combination of ocular and brain lesions is explained by the fact that the retina is embryologically but an outgrowth of the developing brain, and so, being immature nerve tissue, is susceptible to the attack of the organism. In adult toxoplasmosis the central nervous changes are minimal. Parasites are more difficult to find, being present in smaller numbers. Calcification is not found in adult cases of the disease.

Pathological Changes in the Viscera.

Examination of the lungs of children suffering from the disease shows mild interstitial inflammation involving the peribronchi and perivascular tissues. The infiltrating cells are lymphocytes, plasma cells and large mononuclear cells. A little exudate is noticed in the bronchi, but necrosis is rarely seen.

In the heart myocarditis is found, with focal infiltration of lymphocytes, plasma cells, mononuclear cells and eosinophile cells. In the adult the heart is nearly always involved.

In the blood vessels infiltration of vascular cells with lymphocytes and mononuclear cells is found. In foci of acute inflammation polymorphonuclear leucocytes and eosinophile cells are present in large numbers. Parasites are seen in the endothelium of the arterioles and venules.

In the skeletal muscle myositis due to focal infiltration necrosis is present. Parasites have been identified in the muscles.

In the liver, spleen and lymph nodes granulomatous foci are found, with necrosis mainly in the adult. Lesions have been observed in the bone marrow, kidneys, ovaries, testes and subcutaneous tissues.

The cerebro-spinal fluid is xanthochromic, with increase of cells, especially mononuclear cells. The protein content is high.

The ventricular fluid has a high protein content.

The persistent proliferation of toxoplasma in the central nervous system has been explained by the poor diffusion of the specific antibody into the brain substance. In rabbits the ratio of the specific antibody in the serum, brain and cerebro-spinal fluid is as 300:3:1. In the serum and cerebro-spinal fluid this parallels the protein content of tissue fluids—600:20, in milligrammes per 100 millilitres. This explains the apparent "blood-brain barrier" on the basis of the differential diffusion gradient for protein.

The high protein content of ventricular fluid is due to the plasma permeation from damaged vascular tissue walls and in part from the necrotic brain that desquamates into the ventricles. The protein content of the subarachnoid fluid is much lower, owing to the drainage and vascularization of the meninges.

Radiological Diagnosis.

Diffuse intracerebral calcification is the most striking radiological evidence of infantile or congenital toxoplasmosis. The calcification occurs in small flecks and curvilinear streaks, most commonly in the cerebral hemispheres, the parietal, occipital and frontal areas, the basal ganglia and the thalamus. Evidence of increased intracranial pressure is noted in some cases. Pneumoencephalography reveals considerable internal hydrocephalus.

In adults, acute pneumonitis is radiologically the outstanding feature. Acute pulmonary congestion with œdema of the lungs is present, with accentuation of hilar shadows and increase in size and number of the lung markings, radiating outwards from the hilar regions into both lungs, but stopping short of the parenchymal structures. In addition there is the blotchy, blurred appearance seen in acute œdema of the lungs. The picture at this stage is one of passive congestion with pulmonary œdema, but without cardiac enlargement. In advanced cases the picture changes. The accentuated lung markings become subordinated by the increased interstitial infiltration and alveolar exudation. The composite picture is one of conglomerate areas of infiltration, giving the appearance of irregular blotchy consolidation of the lungs. The greatest involvement is in the lower lobes and is similar to atypical pneumonitis or mycotic infections. It may be noted that cerebral calcification in persons suffering from toxoplasmic chorioiditis is never seen if the age of onset can be proved to be over fifteen years.

Diagnostic Tests for Toxoplasmosis.

Skin Test.

The skin test is a hypersensitivity test depending on the patient's reaction to suitable dilutions of the antigen, toxoplasmin, given intradermally. The antigen is derived from the peritoneal exudate of an infected mouse. It is diluted and centrifuged to break up the cell bodies of the toxoplasma. An amount of 0·1 millilitre of antigen is injected into the forearm, and a positive result depends on the development of an area of erythema and induration over 10 millimetres with negative control in twenty-four to forty-eight hours.

This is a crude test and is best suited for big surveys. Of apparently normal people 20% give a positive reaction.

Rabbit Neutralization Test.

The rabbit neutralization test depends on the capacity of human serum to modify the skin lesion in a rabbit's skin. The back of the rabbit is shaved and divided into areas. Toxoplasma from infected mouse-brain suspension in varying dilutions is injected subcutaneously into the rabbit. Controls are used. Skin lesions diminishing in size with increasing dilutions develop in four to seven days. When these varying dilutions of toxoplasma-containing tissue suspension are mixed with suspected human serum some fail to modify the skin lesions and are said to be without antibody. Serum which modifies the skin lesions only slightly has been regarded as yielding equivocal

results of uncertain significance. The serum which completely inhibits the skin lesions and modifies the sizes of others has been regarded as containing toxoplasmic antibodies. The suspected serum must not be allowed to reach room temperature, as it is heat-labile; also, if the serum is diluted the antibody effect disappears. It has been found that 10% of humans without history relative to toxoplasmosis have serum with some antitoxoplasmic properties. There is no way to differentiate between these people and those who have been infected.

Complement-Fixation Test.

The complement-fixation test depends on the fact that toxoplasmic serum contains a substance which in the presence of toxoplasmic antigen has the power of fixing a measured amount of complement. The antigen is derived from the peritoneal exudate of an infected mouse or from toxoplasma-infected chorio-allantoic membrane. The material is frozen and thawed to break up organisms and centrifuged. The antigen is in the supernatant fluid. This in varying dilutions is titrated against a known positive antiserum in certain dilutions. The antigen is used which in the highest dilution gives a "++++" fixation of complement with the highest serum dilution. Complement comes from pooled guinea-pig serum frozen in carbon dioxide. To dilutions of the patient's serum are added antigen and complement. The complement-fixing antibodies are not so heat-labile as neutralizing antibodies. A titre of 1 in 16 is a positive result.

Slide Neutralization Test (Dye Test).

The slide neutralization test (dye test) depends on the ability of serum to modify the staining properties of living toxoplasma; a cytoplasm-modifying antibody is present, which is heat-stable. This is a quantitative test elaborated recently. An accessory factor is present in all human serum in very small amounts, so that even the slightest dilution (1:2) is sufficient to reduce it below effective level. The serum of a suspected person can be transported without refrigeration, as the antibody is heat-stable. The suspected serum is prepared in varying dilutions, and to these is added a suspension of toxoplasma (from the peritoneal exudate of an infected mouse) in fresh, undiluted, normal human serum, which is free of toxoplasmic antibodies, but which contains the important accessory factor. The normal human serum which is the source of the accessory factor can be stored frozen in dry ice and used whenever needed. These mixtures are incubated in a water bath at 37° C. for one hour. Small drops from each tube are mixed with alkaline methylene blue, and the percentage of extracellular toxoplasma whose cytoplasm remains unstained is determined after the mixture has stood for ten minutes. The dye test refers to the highest original dilution of a specimen of serum which, in the presence of a constant, adequate amount of accessory factor, is capable of depriving the cytoplasm of 50% of the toxoplasma of its affinity for the methylene blue at pH 11·0. Tests with experimental animals and human beings with proved recent infections yield serum with dye-test titres of 1 in 1000 to 1 in 16,000. Such serum in the old rabbit neutralization test could not be differentiated from "normal" human serum with toxoplasmic antibodies. However, the dye test result in such "normal" human serum rarely exceeded 1 in 64. It has been found in experimental animals that dye-test antibodies appear in three to five days after inoculation with toxoplasma and reach peak titres of 1 in 1000 to 1 in 4000 in ten to twelve days. The complement-fixing antibodies appear later and can still be absent three to four weeks after infection. Both antibodies have been found to persist for years in high titres.

It is not possible to say that a high titre of antibody is indicative of active infection. The dissociation between the two types of antibodies during the early stages of toxoplasmic infection may, perhaps, provide the only serological means by which activity can be detected. If serum has a high titre of dye-test antibody with little or no complement-fixing antibody—the latter, however, increasing in titre in subsequent tests—there can be a reasonable basis for suspecting an active infection during that particular interval of time.

Persistence of Placentally Transmitted Toxoplasmic Antibodies.

Dye-test and complement-fixing antibodies are transmitted to a normal child and persist in almost undiminished concentration for the first six weeks of life. There is a gradual diminution of antibodies from then on, but the dye-test antibodies, being able to be detected in higher dilutions, are still present in small amounts at the end of nine months. In children who were proved to have congenital toxoplasmosis by quantitative serological methods it was found that the antibodies persisted in undiminished or even higher concentrations.

Isolation of Toxoplasma.

In the acute stage the organism may be recovered from the blood, from the bone marrow by splenic puncture, from the cerebro-spinal fluid and possibly from the sputum. In the subacute stage it may be recovered in the ventricular fluid and central nervous system at autopsy. In the chronic stage it may be recovered from the central nervous system, the eye and the myocardium.

Examination of direct smears is successful only from aspirated ventricular fluid sediment. Animal inoculations are necessary to study the biological characteristics. Several species of animal should be used, as toxoplasma is common in animals.

To isolate toxoplasma, use an inoculum such as ventricular sediment and inject it into mice and guinea-pigs. Inoculate half intracerebrally and half intraperitoneally. If the animal dies, remove the brain aseptically and subinoculate it into other animals. Impression films are prepared and examined for the organism. The intraperitoneally inoculated animals are examined for peritoneal exudate on the sixth to tenth day. The aspiration of the peritoneal exudate is carried out with a syringe from lightly anæsthetized animals. The pooled fluid is removed under aseptic conditions and is subinoculated intracerebrally into a fresh group of mice and guinea-pigs. If these animals die, take impression films from the liver, spleen and lungs, and look for toxoplasma. If none die, then kill half in one month, pool the brains and subinoculate them. This "blind passage" may bring to light a toxoplasmic infection. The other half are then tested for immunity if organisms are found.

A distinction has to be made from *Histoplasma capsulatum*, *Trypanosoma cruzi*, leishmania, encephalitozoon and sarcocystis.

Treatment.

No specific treatment is known. Some reports state that there has been improvement with sulphonamides and penicillin.

A syndrome in infancy has been described, characterized by extensive brain tissue destruction with hydrocephalus, cerebral calcification and chorio-retinopathy associated with bizarre "degenerative" changes in small blood vessels, which can be distinguished from toxoplasmosis only by means of serological tests or pathological examination. With regard to the role of intracerebral calcification and chorio-retinopathy in cases of toxoplasmosis, it has been demonstrated that when infantile chorio-retinopathy is associated with positive quantitatively significant results to serological tests for toxoplasmosis, the incidence of grossly perceptible cerebral calcification in X-ray films of the skull was 90%. In children with chorio-retinitis and negative serological findings, the incidence of cerebral calcification was only 5%.

Report of a Case.

J.K., a male, aged twenty-eight years, married, with no family, consulted me on account of defective vision. The history taken from the patient's mother revealed that at the age of six months the child had been noticed not to follow objects, and the eyes appeared to wander. The child was taken to a medical man, but I was unable to learn his name to refer to his clinical notes. The mother states that the doctor said that the sight would improve with time. At the age of five years the patient was examined at the Eye and Ear Hospital in Melbourne, where the mother was told that the sight was defective and that nothing could be done

to improve vision. The mother states that her confinement was normal, and that she had had no illnesses during pregnancy. No history of convulsions in the patient as a child was obtained. The family history was clear. I examined the mother and found no ocular abnormality, and an X-ray film of the mother's head revealed no intracerebral calcification. There were five other children, not in Sydney, but the mother states that their eyes are normal. The father of the patient has normal vision. The only pet kept at the home when the present patient was born was a dog, which was stated to be apparently well.

The patient was a man in apparent good health and of average intelligence. The uncorrected visual acuity in the right eye was less than 6/60, and in the left eye 6/36. The patient was able to read "J4" type with the left eye. There was a fine horizontal nystagmus (about 60 per minute), more pronounced in the right eye. The patient fixed objects mainly with the left eye, and the right eye was very slightly depressed. The pupils were equal and active. The extra-ocular muscles had a full range of movement. Correction in the right eye was with a −3·5 cylinder with the axis at 90°, but the visual acuity did not improve. The left eye correction was with a −0·75 sphere with a +1·0 cylinder axis at 160°, the visual acuity in the left eye being improved to 6/24 (one letter only). The visual fields were full when the confrontation test was applied. There was a central scotoma on the Bjerrum's screen, more pronounced in the right eye. The lens and vitreous were clear.

Examination of the fundus of the right eye (see Figure I) revealed a slightly pale disk with considerable pallor in a wedge-shaped area between the exit of the superior and inferior temporal vessels, corresponding to the exit of the papillo-macular bundle. The macular area consisted of a large, elliptical, pigmented hole, measuring about three disk

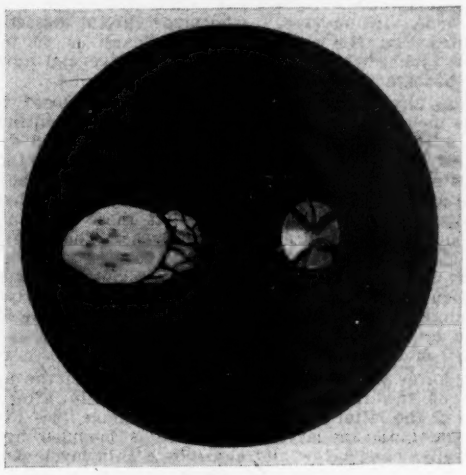

FIGURE I.

diameters horizontally and about one and a half disk diameters vertically. The white sclera could be seen shining at the bottom of the lesion, and here and there chorioidal vessels were observed to give a red contrast. The edge of the lesion was pigmented, and pigment was scattered in small quantity in the area of the lesion. The retinal vessels had a normal exit from the disk, a cilio-retinal artery making its exit from the lateral aspect of the disk and passing in close relation to the inferior edge of the macular lesion. The vessels themselves appeared normal. The peripheral groundwork of the retina was normal.

Examination of the fundus of the left eye (see Figure II) revealed slight pallor of the disk with a small, pale, wedge-shaped area between the exit of the inferior and superior temporal vessels, as in the right eye. The pallor was not so pronounced as in the other eye. Examination of the macular region revealed a roundish, pigmented hole of about one and a half disk diameters. The white sclera and reddish chorioidal vessels were seen in the depth of the lesion. The edge of the lesion was pigmented, and generally the lesion was much more heavily pigmented than its fellow of the right eye. The vessels made a normal exit from the disk, and also a cilio-retinal artery could be seen emerging from the lateral aspect of the disk and coming into close relation with the superior edge of the macular lesion. The vessels appeared normal. The retinal ground was normal except for an area of pigment of about one disk diameter lying at the first branch of the superior nasal retinal artery.

The visual fields were difficult to measure accurately, but were not greatly reduced. There was a bilateral central scotoma, much more pronounced in the right field. No abnormality was found on general examination of the patient.

X-ray films of the patient were taken, antero-posterior (see Figure III) and lateral (see Figure IV). Stereoscopic X-ray pictures revealed intracranial calcification. The calcified areas were discrete, lying in the frontal, temporal and occipital areas of the brain.

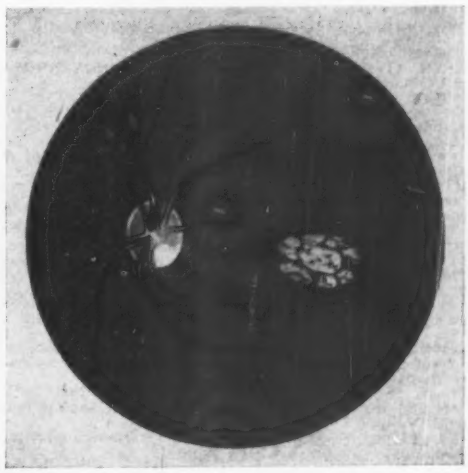

FIGURE II.

With regard to serological investigation of this patient, it may be mentioned that Dr. Harry A. Feldman, of New York, stated in a letter to me that "serological data are usually quite difficult to interpret in adults because so many persons of the patient's age have antibodies for Toxoplasma". In view of this, of the twenty-eight years that had elapsed since the patient's infection, and of the patient's reluctance, no serological investigation was carried out. A negative or positive result would not alter the diagnosis in view of the above-mentioned facts.

Summary.

1. Atypical encephalitis in children should lead one to suspect toxoplasmosis. Examination of the fundi, X-ray examination of the skull and serological investigation should be carried out. If any two of these investigations give positive results, then the disease may be classified as toxoplasmosis.

2. If the infection has subsided at birth, then sequelæ may lead to diagnosis. Bilateral macular chorioido-retinitis, together with X-ray evidence of intracerebral calcification, is very strong evidence of past infection with toxoplasma.

3. In the differential diagnosis of atypical pneumonitis in adults, toxoplasmosis must be included in the differential diagnosis. It may be noted that if the infection occurs after the age of fifteen years, then intracerebral calcification is not found.

4. Acute uveitis for which no cause is found must be suspected of having toxoplasmosis as an ætiological factor.

5. New serological tests have been devised for the detection of the disease in the acute stage.

6. Sabin has recently reported the results of post-mortem examinations on babies with chorio-retinopathy and intracerebral calcification which do not appear to be due to toxoplasmosis. Further work is apparently being carried out to clarify the position.

Acknowledgements.

Thanks are due to the medical artist, Mr. Woodward Smith, for his excellent drawings and photographs, to Dr. A. Colwell for the X-ray reports, and to the authors of various articles dealing with the disease from whose works I have freely drawn.

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THE LARYNGEAL VENTRICLE AND ITS APPENDIX (LARYNGEAL SACCULE) AND THEIR ASSOCIATED PATHOLOGICAL LESIONS, WITH THE REPORT OF THREE CASES OF LARYNGOCELE.

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Anatomy.

THE laryngeal ventricle is a fusiform recess lying in the horizontal plane at the mid-laryngeal level, between the true vocal cord below and the ventricular fold or band (false cord) above. It extends laterally toward the thyroid ala and upwards for a short distance lateral to the ventricular band. It is observed only in mammals.

Its upper and medial wall is formed by the ventricular band, its floor by the lateral portion of the vocal cord and the thyro-arytenoideus muscle, covered by mucous membrane; the lateral wall is formed by the upper fibres of the thyro-arytenoideus lying on the perichondrium of the thyroid cartilage. From the anterior part of the ventricle a narrow slit-like opening leads upwards into the appendix of the ventricle (synonym: *sacculus ventriculi laryngis*), which itself lies between the upper part of the ventricular band medially and the inner surface of the upper part of the ala of the thyroid cartilage laterally.

The sacculus is thought to be the atavistic representation in man of the cervical pouches or air sacs in anthropoid apes. Detailed description of the extra-laryngeal air sacs in monkeys is given by Negus in his "Mechanism of the Larynx" (1948); he mentions also buccal air sacs in frogs and abdominal air sacs in birds. In the anthropoid ape these sacs extend from the ventricle through the thyro-hyoid space and membrane into the neck; the main sac is usually globular in shape, but extensions may pass up to the angle of the jaw and down near the mid-line of the neck on to the chest and laterally under the pectoral muscles (see Figure IX). They are said to serve as reservoirs for a supplementary supply of air to be used without interruption of, or interference with, the fixation of the thorax for the independent use of the forelimbs. In the howler monkey the sac has rigid light osseous walls. The possible relation between the appendix of the ventricle or sacculus and these air sacs in certain monkeys is further discussed in the section dealing with laryngocele (*quod vide*). The sacculus varies greatly in size and shape; sometimes it is nearly conical with its base below, sometimes pear-shaped with the broader part above, at other times almost cylindrical, and it is usually slightly curved upon itself in a backward direction. It measures about half an inch in the vertical direction, usually extending almost to the upper border of the thyroid ala; very rarely it extends as high as the hyoid bone. Hilton (1837), who made careful dissections, estimated that its walls contain as many as 60 to 70 mucous glands which lie in loose connective and adipose tissue beneath the mucosa.

Hilton also found that the sacculus is enclosed in a fibrous membrane or capsule. This capsule is continuous below with the ventricular band or false cord and firmly attached to it along its anterior half or even two-thirds. The anterior aspect of this capsule is attached to the epiglottis and its superior and lateral aspect to the thyroid cartilage. It was Hilton's belief that these attachments were the chief safeguard against prolapse. The lateral or thyroid ala surface of the sacculus is covered in its upper part by the superior fibres of the thyro-epiglottideus muscle, which curves upwards and inwards at first posteriorly and then slightly anteriorly around it to blend with the fibres of the *aryteno-epiglottideus inferior* (*compressor sacculi laryngis* of Hilton) described below.

The lower lateral part is covered by some of the upper fibres of the thyro-arytenoideus immediately after the origin of the latter from the thyroid cartilage (see Figure I). The inner or laryngeal surface is bounded by the ventricular band below and above by a thin layer of muscular fibres the *aryteno-epiglottideus inferior*, which is the inferior part of the same muscle found in the aryteno-epiglottic fold (Figure II). It crosses the summit of the pouch from behind forwards and a little on its medial aspect. Hilton thought that these muscle fibres compressed the sacculus, and caused the secretion of its glands to discharge upon the vocal cords and so lubricate them. However, it is more likely that the whole muscular envelope, just described, by concerted or separate action will have this effect on all the glands or different sections of them at different times according to the variability of action of the separate muscles.

The mouth of the sacculus is situated in the anterior third of the roof of the ventricle, and is of narrow oval shape about eight millimetres average length in its longest diameter, but is said to be more circular if the cords are relaxed. Just below the ventricular orifice of the sacculus is a delicate horizontal fold of the mucosa extending medially from the lateral or thyroid ala wall.

Hilton described two crescentic folds placed anteriorly and posteriorly at slightly different levels, the anterior being the higher; but Irwin Moore (1922) found that there was considerable variation of the minute details (Figure III).

The ventricle and sacculus are lined by ciliated columnar epithelium. As well as the saccule, the ventricular band on the medial wall of the ventricle is also rich in mucous glands in the *tunica propria*, whereas the lateral and lower walls of the ventricle are much less well supplied. In this lateral wall the mucous membrane is often thrown into a fold which may become hypertrophied, and in certain pathological states—for example, in tuberculous infection of the larynx—project across the ventricle beyond the lower border of the ventricular band or false cord and, as was pointed out by Lennox Browne as long ago as 1899, so afford a reasonable explanation of the condition described as eversion or prolapse of the ventricle, or produce a condition which simulates it (Figures XV and XVI). There has always been considerable doubt as to whether this condition is caused as its name implies. Albrecht (1896) found in the dog that a fold was present on the lower part of the lateral wall of the ventricle, lying opposite the thyro-arytenoid muscle and possibly raised up by it. This fold is less pronounced anteriorly than posteriorly. Experimentally caused hyperplasia of this band produced a condition simulating prolapse of the ventricle in man.

FIGURE I.

Larynx with right ala of the thyroid cartilage removed, showing the external or lateral aspect of the right *sacculus laryngis* with its associated glands and the relationship of the thyro-arytenoid muscle to it. Originally drawn from a dissection by John Hilton (after Irwin Moore).

Pathology of the So-called Prolapse of the Laryngeal Ventricle.

So-called prolapse of the laryngeal ventricle (Figures IV to VIII) is rare, Irwin Moore (1922) tracing only 82 recorded cases up to 1922. There is considerable doubt whether true eversion ever occurs except in association with traction of a cyst or tumour. Shattock's (1922) hypothesis (enlarged upon in the section dealing with eversion of the sacculus *quod vide*) is that negative pressure produced by the blast of air liberated above the cords during the act of coughing, in association with relaxation of the mucosa of the ventricle as a result of a chronic pathological state, may possibly produce a true eversion. Compare this hypothesis with that of an hypertrophied mucosal fold in a chronically inflamed larynx, as suggested by Lennox Browne and Albrecht and described above.

Mucosal or subepithelial retention cysts and neoplasms may produce eversion by the displacement which the growth causes (Figure VIII) or by traction due to their weight.

Moore points out that if eversion of the sacculus occurs by a similar mechanism, true eversion of the ventricle may slowly follow—a process of turning inside out. Fletcher Ingals (1899) recorded a case in which this apparently occurred (Figure VIII).

Moore studied the 82 cases of so-called prolapse of the laryngeal ventricle he found recorded in the literature up to 1922. In the 82 cases, laryngeal tuberculosis was present in only three and syphilitic lesions of the larynx in two (and the infection was by no means certainly causative in any of these five). Tumours accounted for five cases (four benign and one malignant), cysts for five, and of the remaining 57 cases, chronic laryngitis was present in the majority. He concludes that beyond the inflammation and ulceration to which tuberculosis and syphilis give rise with their accompanying coughing, there is no evidence that they play any special part in the production of so-called prolapse. There was no clear example in this series in which simple eversion of the ventricular wall was unaccompanied by an inflammatory swelling. In other words, he holds the belief that even in the case of a cyst or benign neoplasm which might be expected to cause an eversion by its size and weight, there must be an inflammatory factor such as acute or chronic œdema or hyperplasia weakening the attachments of the ventricular walls as well. In my opinion the negative pressure associated with coughing (the latter itself nearly always an accompaniment of acute and chronic laryngitis), as suggested by Shattock, is probably an additional important factor. Direct trauma to the larynx, or indirect trauma such as excessive vocal strain, may rarely be another factor.

On indirect laryngoscopy the observer sees a rather elongated or pear-shaped smooth, firmly œdematous-looking tumour; the mucosa over it is very similar in colour to the adjoining mucosa and distinct from that on the true cords. It can be seen protruding between the true and false cords and covers part or even the whole of the true cords. The tumour can be partly or wholly replaced into the ventricle on direct laryngoscopic examination, but protrudes again immediately when the patient coughs.

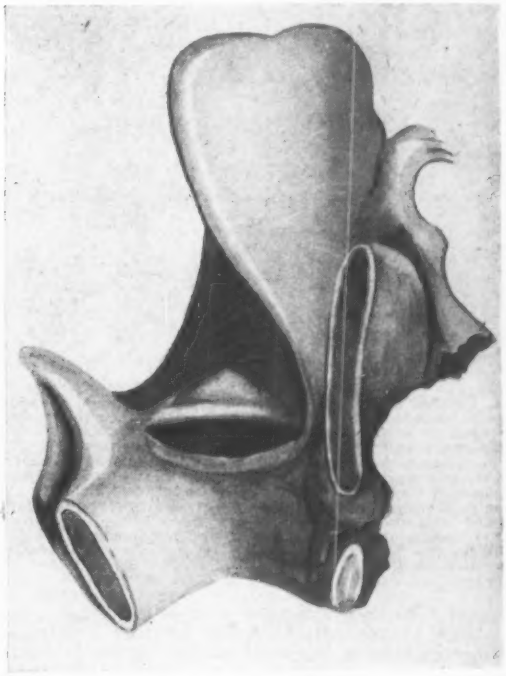

FIGURE II.

Section through the right side of the thyroid and cricoid cartilages, showing the left ventricle of the larynx, the left *aryteno-epiglottideus inferior* muscle (*compressor sacculi laryngis* of Hilton) and a triangular area of the inner wall of the sacculus (exposed by removal of mucous membrane) below this muscle but above the ventricular band. This is the weak area through which a hernia of the sacculus can develop. Originally drawn from a dissection by John Hilton (after Irwin Moore).

Naturally different tumours of this type vary in shape and size depending upon ætiology and age. The size may vary from a fold of tissue up to that of a small cherry or larger. The larger ones interfere with phonation.

They have been described as tapering anteriorly, the posterior extremity being more rounded and the upper surface flattened. The anterior and posterior borders of the tumour are said always to fall sharply to the level of the vocal cord. If this latter observation was always true, it would constitute a helpful diagnostic finding. Other variations are dependent on the ætiology and on the presence or absence of cyst formation. In diagnosis, new growths benign and malignant may be mistaken for this condition (mainly the former), and because of the size, colour, smooth surface, consistency and absence of a pedicle the fibroma group especially.

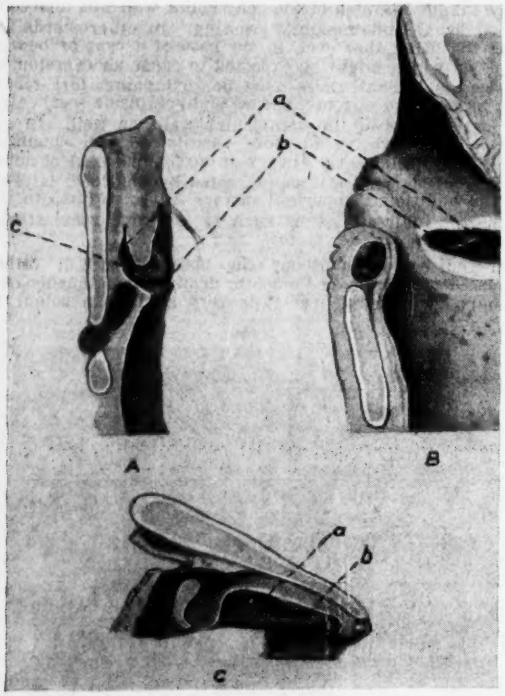

Figure III.

A. Coronal section of larynx through the middle of (*a*) sacculus and (*b*) ventricle, showing the opening of the former into the latter, and (*c*) a horizontal fold of mucosa. *B*. Posterior half of same larynx viewed from the inner aspect, showing (*a*) opening of the sacculus, (*b*) posterior end of ventricle, and between them the posterior frænum of Hilton. *C*. Horizontal section of right half of same larynx, carried between the ventricular band and the vocal cord (seen from below), showing (*a*) mouth of sacculus, (*b*) cut edge of mucosa which marks the limit of the ventricle. Originally drawn from a dissection by S. G. Shattock (after Irwin Moore).

Microscopic examination of a piece of tissue removed at biopsy is usually the final arbiter (Figure VI). In simple eversion of the sacculus the protrusion is confined to the anterior part of the elongated ventricular opening.

The differential diagnosis from tuberculous and syphilitic granulomatous lesions is made on clinical grounds taken in conjunction with the result of radiographic examination of the chest and serological tests.

With regard to treatment, most of these lesions cause little inconvenience, in which case no treatment is necessary. Dyspnœa and stridor may necessitate the removal of the tumour. In acute cases, rest and the use of a vasoconstrictor spray and inhalations during the stage of acute laryngitis usually result in the disappearance of the tumour when the acute stage has passed.

In the more chronic cases, daily replacement of the tumour with a probe followed by the application of astringents may be successful, but if not, and if symptoms are annoying, removal is indicated. Diathermy cautery, the cold wire snare and punch forceps have been used according to the type of case. Laryngofissure may be necessary for good access. If the correct amount is taken, the scarring or healing will cure the prolapse. If too much is removed, contraction may make a concave edge to the cord or interfere with arytenoid movement. Prolapse of the ventricle or eversion of the sacculе or both combined will ultimately cause permanent *myasthenia laryngis*, and alterations in the laryngeal mechanism that will leave a permanently impaired voice, even after removal of the dislocated mucosa (Jackson, 1947).

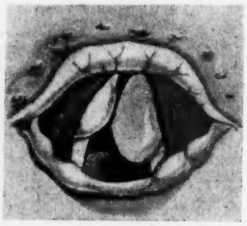

Figure IV.

Prolapse of both ventricles of the larynx (Leffert's case, after Irwin Moore).

Pathological Lesions of the Sacculus Ventriculi Laryngis.

Pathological lesions of the *sacculus ventriculi laryngis* may be classified as follows: (i) laryngocele, (ii) hernia of the sacculus, (iii) eversion of the sacculus, (iv) tumours of the sacculus—(*a*) cystic, (*b*) solid.

Laryngocele.

The term "laryngocele" (synonym "aerocele of the larynx") was introduced by Virchow in 1867 to describe thin-walled air sacs which arise from the laryngeal ventricle, including of course the sacculus. He made the following statement:

> I have found a dilatation of the ventricle of Morgagni, up to the present unknown as it seems, which one may call "ventricular laryngocele". One sees, in these cases, small elongated sacs with thin walls, coming off from the upper part of the ventricles by a somewhat narrow orifice and sometimes reaching to the upper border of the thyroid cartilage, and even to the hyoid bone, where they terminate by an end in the form of a club; most frequently they are found only on one side; once, however, I have met them on both sides. Their walls are smooth, their interior is lined with ciliated epithelium and they are filled with air.

There are apparently two groups of laryngoceles, the grouping being based on ætiological grounds, and furthermore, in some cases the two ætiological factors are combined.

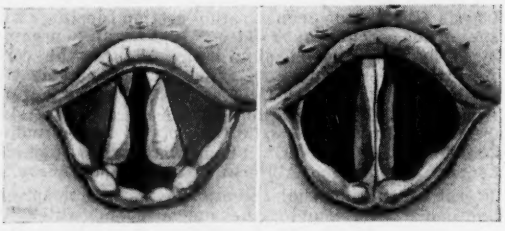

Figure V.

Prolapse of both ventricles, left during respiration and right on phonation (Elsberg's case, afer Irwin Moore).

In the first and less common group the extra laryngeal air sac in the neck is an atavistic remnant of the air sacs which serve the anthropoid apes as supplementary reservoirs for respiratory air, and which are described above in the section dealing with anatomy. This type is more

likely to be bilateral and symmetrical. The sacs are lined by ciliated columnar epithelium, and lymph nodes are found in the subepithelial tissue as in branchial cysts.

In the second group, the air sac would appear to be caused by a herniation of a portion of the laryngeal wall into the tissues of the neck as a result of excessive pressure, either below a pathological stricture at the level of the false cord, or because of the repeated effect of such an excess pressure below approximated false cords. When there is stenosis of the larynx with scarring and distortion of the true cords, phonation is carried out either wholly or partly with the false vocal cords. The hernia may occur in the lower part of the larynx and appear through the crico-thyroid membrane. A similar herniation may very rarely occur between the rings of the trachea, and examples of tracheocele have been recorded. In these cases, trauma or a local pathological lesion has usually caused a predisposing weakness. More commonly, though still rarely, the herniation takes the form of a dilatation of the sacculus which presents through the thyro-hyoid membrane. A congenitally long appendix of the ventricle (or sacculus) may be of ætiological significance, and in this way the two factors of excess pressure and atavism may be combined.

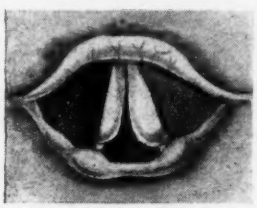

FIGURE VI.

Œdematous hyperplastic folds of ventricular mucosa simulating prolapse of the ventricles (Chiari's case, after Irwin Moore). Chiari removed these piecemeal and decided their identity after histological examination.

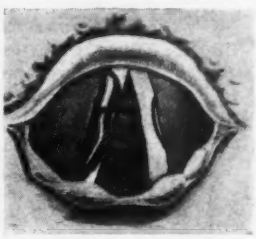

FIGURE VII.

Traumatic laryngitis; submucous hæmatoma following blow on right side of larynx with prolapse of right ventricle. (Abraham's case, after Irwin Moore.)

In support of the hypothesis that many laryngoceles are herniæ of the larynx, it is recorded that Larrey (Blewett, 1939)—variously spelled as Larrey, Larrez, Larney—who was physician and surgeon to Napoleon's armies in Egypt, observed these air sacs in blind Arabs employed by the priests to chant the verses of the Koran from the minarets of the mosques. He also found a similar condition in a drill instructor. Jackson (1947) thinks it is not so rare and that it is often overlooked. Apparently in conditions in which there is an obstruction it often develops. Lindsay (1940) describes occasions on which the false cords close sphincterically; one of these is just before loud sounds are produced. This causes excess pressure in the ventricles, hence hernia of the saccule in an external direction may occur in singers; it may occur after violent crying, sneezing or even nose blowing, and in those who play wind instruments, especially horn-blowers. Once the herniation has occurred, there is often a check valve action at the narrow bottleneck in the thyro-hyoid membrane; air passes into the sac and cannot escape so easily and pressure builds up. This has been proved by X-ray examination including planigraphic studies (Jackson, 1947). This type of sac is often not lined by mucous membrane, only by fibrous tissue; but it may be lined by flattened epithelium or even by ciliated epithelium which has grown in from the sacculus. However, lymph nodes are not found in the *tunica propria*.

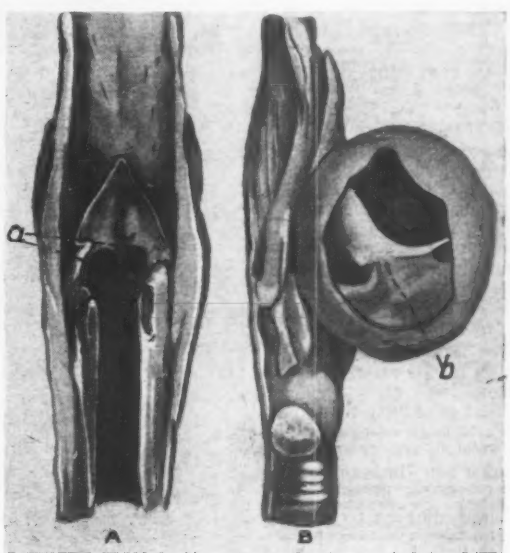

FIGURE IX.

Hylobates syndactylus (gibbon). In this species of monkey there is a fusion of the sacculus on each side to form a single large air sac in the mid-line of the neck. A, posterior view showing (*a*) opening of the ventricles. B, lateral view of the air sac, showing its partial subdivision by a median septum (*b*).

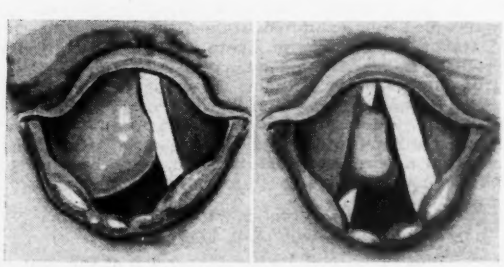

FIGURE VIII.

Eversion of the right ventricle of the larynx secondary to a cyst of the saccule, which extended up into the right vallecula and down into the larynx (Fletcher Ingal's case, after Irwin Moore). Left, before aspiration; right, after aspiration, showing the everted ventricular wall.

In the type of laryngocele extending from the sacculus there is a narrow connexion with the cavity in the side of the neck, either through the thyro-hyoid membrane, or very rarely posterior to the lateral ala of the thyroid cartilage. Judging from the reported cases, in the former common type the pedicle may pass through the thyro-hyoid membrane either close to the mid-line or more laterally (Figure X). In the neck, a soft elastic swelling is found which bulges during coughing. Again, after a study of the reported cases, it would appear that three types of laryngocele are found clinically in relation to the sacculus. In the first there is simply a soft elastic air-containing swelling in the neck, which increases in size on increased expiratory pressure, and there are no intralaryngeal signs (Figures XVIIIA and B).

In the second group mentioned by Lindsay (1940) an internal laryngocele develops which extends to the aryepiglottic fold or even to the base of the tongue, occupying one vallecula. This is the hernia type which dilates the sacculus in an upward direction instead of out through the thyro-hyoid membrane. This type may bulge into the larynx at its lower extremity, as described in relation to the third or combined type.

In the third type there is an external cavity as described in relation to the first group, but there is also an internal swelling, which protrudes into the lumen of the larynx simulating prolapse of the ventricle (see Figure XVIIA), and at the same time distends the ventricular band. It can be described as the combined type (Figure XVIIB). Jackson states that in these cases (which Lindsay describes as combined internal and external laryngocele), pressure on the neck sac enlarges the intralaryngeal sac and *vice versa*, and also that the condition may be associated with eversion of the ventricle. I incline to think that this latter is common or almost invariable in this class of case.

There must be a check valve action other than at the level of the thyro-hyoid membrane for this class of laryngocele to develop —possibly at an over-stretched slit-like normal opening between the ventricle and the sacculus. If the intralaryngeal protrusion is very large, a complaint of hoarseness or even dyspnœa may be made.

A tympanitic note can be obtained on percussion if the neck sac is large. Sometimes a hissing or squealing sound (if the mouth is open) is heard if pressure is made on the neck sac and the air is forced into the larynx.

The larger neck sacs are as a rule relatively thin-walled and the smaller ones have thick walls, though of course all stretch and become thinner on increasing expiratory air pressure.

The diagnosis of laryngocele rests upon the clinical findings described and is confirmed by X-ray examination; this reveals an air-containing cavity which, as a rule, has a tapering neck in the region of the thyro-hyoid membrane. Infection of the cavity sometimes occurs, and only then may attention be drawn to it because of increase in size and tenderness. A dependent fluid level is then seen on X-ray examination (Figures XVIIIA and B), and an offensive smell or taste may be noticed in the throat when the neck swelling is pressed. Frank pus may even be coughed up. The uncomplicated laryngoceles require no treatment; if they are infected excision is indicated. The type of case in which there is only an external sac presents no difficulty. Lindsay, in discussing the combined type, describes removal of the external sac and then splitting of the thyroid ala just in front of the inferior horn for access in removal of the internal sac. However, this latter manœuvre may never be necessary. In one of Jackson's cases of the combined type, removal of the intralaryngeal protrusion caused a disappearance of the laryngocele; there was no trace of it at all, or at least it was not demonstrable clinically three years later. This may have been because the check valve mechanism, which has a progressive action to increase the size of the combined type, had been by-passed and eliminated by the action of perforating the intralaryngeal sac.

FIGURE X.

Larynx showing right *sacculus ventriculi laryngis* extending through the thyro-hyoid membrane anteriorly and lying on the lateral surface of this membrane (Gruber's case, after Irwin Moore).

The condition of laryngocele is so uncommon that any one observer could not hope to see many examples in a lifetime; but in the hernia type one would expect the pedicle to pass through the thyro-hyoid membrane at its weakest spot, and this could be where the internal laryngeal nerve and vessels pass through. In the true atavistic type, a path is already present at birth, and on analogy with the sacs in monkeys the pedicle may well be found nearer the mid-line.

Hernia of the Sacculus Laryngis.

Hernia of the *sacculus laryngis* is a distinct pathological lesion and is non-developmental in origin.

Increased expiratory pressure causes a medial protrusion of the upper wall of the sacculus at the weak spot between the lower margin of the *aryteno-epiglottideus inferior* muscle and the ventricular band (Figure II).

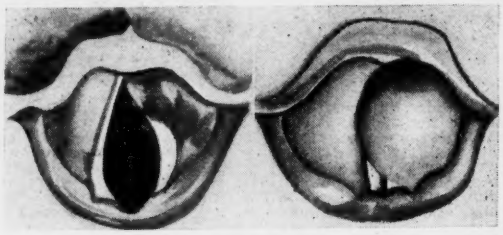

FIGURE XI.

Hernia of the left *sacculus ventriculi laryngis* (F. Spicer's case, after Irwin Moore). Left, on quiet respiration; right, on phonation.

It may eventually involve the whole of the sacculus except its lowest and outer part (Figure XI).

It is especially liable to occur in cases in which for some reason (for example, stenosis of the larynx with scarring and distortion of the true vocal cords) phonation is carried out either wholly or partly with the false vocal cords. The pressure of air beneath the abnormally closed and phonating ventricular bands leads first to inflation of the sacculus via the ventricle, and then as the pressure is increased the herniation takes place medially into the laryngeal airway just above the ventricular band. This takes place suddenly and usually audibly "with a pop"; a tumour the size of a small cherry may appear, of a paler colour than the remainder of the laryngeal mucosa because of its distension.

Several well-authenticated examples of this type have been recorded and exhibited at medical society meetings from time to time; Irwin Moore (1922) fully discusses three—Solis-Cohen (Philadelphia) in 1887, Garel (Lyons) in 1904, and Frederick Spicer (London) in 1921. The condition is akin to the hernial type of laryngocele. It is very rare. Unless the hernial protrusion caused discomfort, treatment would not be necessary. If it had to be removed a permanent fistula could occur by means of which air could by-pass the false cords on phonation.

Eversion of the Sacculus Without (and With) Eversion of the Ventricle.

Eversion of the sacculus without eversion of the ventricle is a very rare condition, but can occur. Moxon (Guy's Hospital, London) in 1868 first recorded a case, and the specimen was still in the museum of Guy's Hospital in 1922 (Figure XII). He found it at the autopsy on a male patient who had died of cancer of the stomach; no laryngeal symptoms were present during life. There was a small pendulous "tumour", something like the outer edge of a small horseshoe in outline, which protruded from the anterior half of the ventricle of the larynx and hung down over the vocal cord. It could be easily inverted and replaced in the normal position of the sacculus.

Over fifty years later, in 1921, the specimen was re-examined by Professor Shattock of the Royal College of

ILLUSTRATION TO THE ARTICLE BY F. H. MILLS, J. K. MADDOX, R. B. BLACKET, AND A. JEAN PALMER.

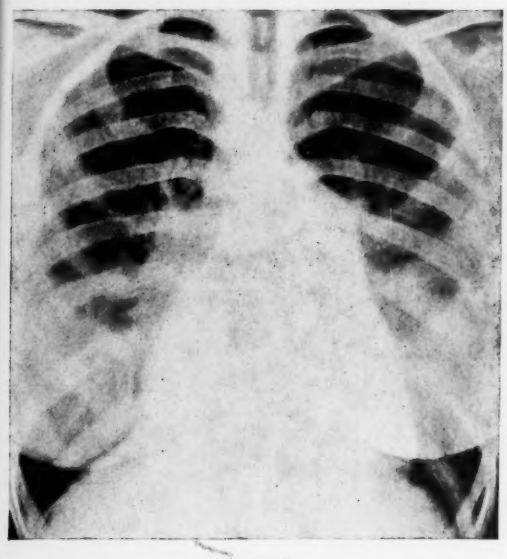

FIGURE I.
Postero-anterior view of chest in Case II, to show pulmonary venous congestion and hæmosiderosis, dilatation of pulmonary artery and left auricular enlargement.

ILLUSTRATION TO THE ARTICLE BY RICHARD FLYNN.

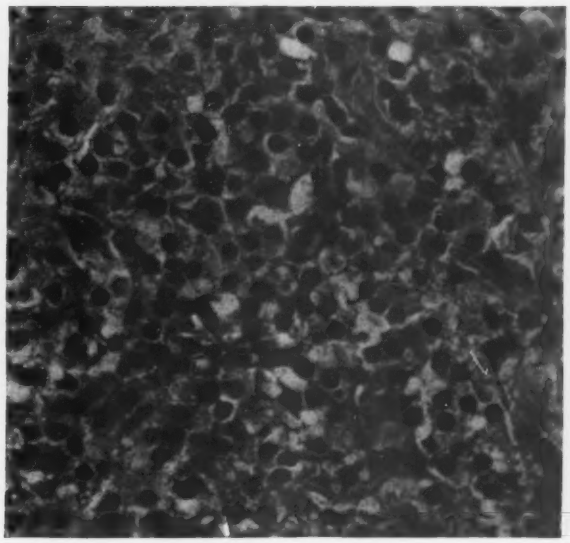

FIGURE I.

ILLUSTRATIONS TO THE ARTICLE BY IAN B. JACK.

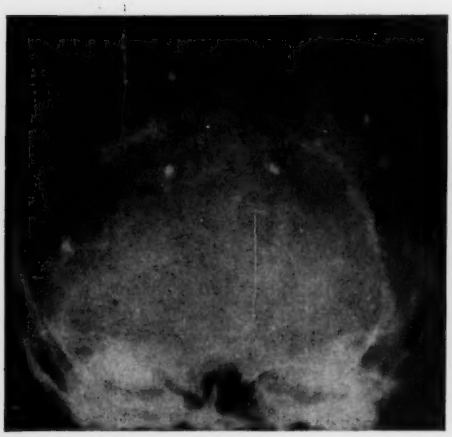

FIGURE III.

FIGURE IV.

ILLUSTRATIONS TO THE ARTICLE BY GEORGE SWINBURNE.

FIGURE XVIIIA.

FIGURE XVIIIB.

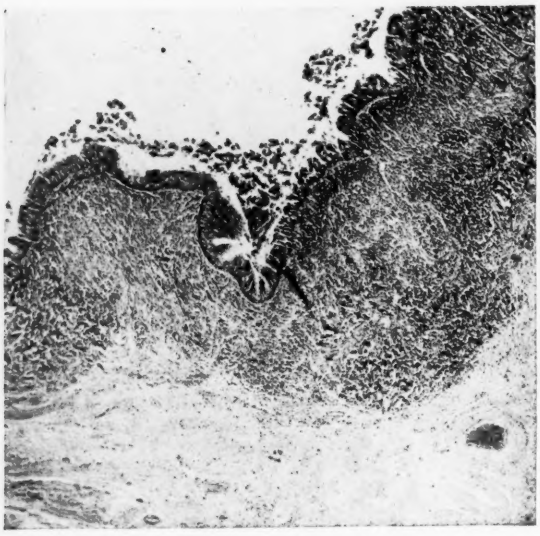

FIGURE XXA.

FIGURE XXB.

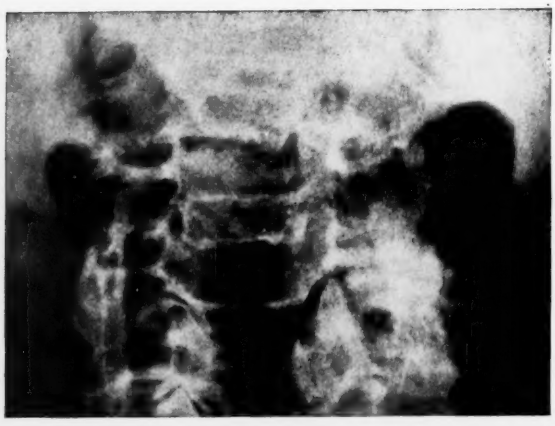

FIGURE XXII.

FIGURE XXIII.

Surgeons, and by Irwin Moore, who removed a portion of the left thyroid ala and found that the "tumour" was indeed the sacculus, as it was absent from its usual position when the "tumour" was down and it could be replaced.

Morell MacKenzie in 1871 also recorded a specimen which was in the museum of Golden Square Hospital (Figure XIII) and which was also reexamined by Professor Shattock and Irwin Moore in 1921 and found to be a true specimen of everted sacculus similar to Moxon's (Figure XIV). There was a similar but less pronounced eversion of the sacculus of the opposite side in this specimen.

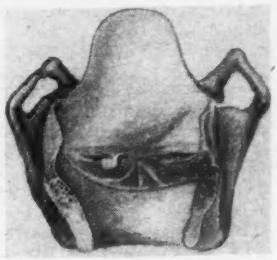

FIGURE XII.

Eversion of the left *sacculus ventriculi laryngis* (Moxon's case, after Irwin Moore), originally drawn by W. T. Shiells from the specimen in the museum of Guy's Hospital, London.

A spurious eversion of the sacculus may occur from a hyperplastic inflammatory condition of the mucosa of the laryngeal ventricle and sacculus, usually of tuberculous origin. The condition is really papillary hyperplastic tuberculosis, and flattened leaf-like processes arise from the walls of the cavities and may extend downwards and medially so far as to protrude into the laryngeal cavity (Figures XV and XVI). This condition has been mentioned in the section dealing with prolapse of the ventricle of the larynx.

There has been considerable discussion regarding the cause of true eversion of the sacculus other than the type associated with a cyst or neoplasm which drags upon the lining of the cavity. In the latter case eversion of the ventricle also may occur.

In regard to the type described by Moxon and Morell MacKenzie, Nolan Mackenzie (1882) expressed the opinion that perisaccular inflammation accompanied by hyperplasia of the *tunica propria* with fatty degeneration and wasting of the surrounding muscles were prerequisite to a true eversion. The fibrous sheath holding the sacculus in place is weakened and with the walls of the sac may sag downwards towards the cavity of the larynx.

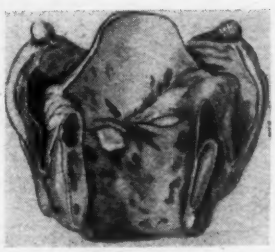

FIGURE XIII.

Eversion of the left *sacculus ventriculi laryngis*; partial eversion of the right sacculus (Morell MacKenzie's case, after Irwin Moore). Originally drawn by W. T. Shiells from the specimen in the museum of the Hospital for Diseases of the Throat, Golden Square,

Shattock, in 1921, after studying this subject, thought that negative pressure caused by violent coughing was the main factor. In all probability both the above-mentioned factors help to produce the condition. The perisaccular inflammation may cause a loosening or flabbiness of the mucosa with its glands in relation to the deeper areolar tissue, and the negative pressure probably supplies the force necessary to cause the eversion.

Shattock, at the December, 1921, meeting of the Section of Laryngology of the Royal Society of Medicine, produced a model he had made and demonstrated his hypothesis. He thinks that when the glottis is suddenly opened in coughing, the blast of liberated air rushes by the ventricle and orifice of the saccule, producing a negative pressure which may lead to some degree of loosening of the attachments of the sacculus and then to its complete eversion. He mentioned as another factor the drag following forcible attempts to dislodge an unusual amount of stringy mucus secreted by the saccular glands and hanging down towards the true cords, but this is unconvincing. Shattock's model consisted of a length of rubber tubing of the calibre of the trachea with a horizontal oval slit cut in its side. Over this slit a short piece of the blind end of a rubber finger stall was fixed. Each time the tube was blown through the finger stall collapsed, being drawn inwards by the negative pressure so produced.

FIGURE XIV.

The same specimen as in Figure XIII with the everted *sacculus laryngis* replaced into its normal position and lightly distended with cotton wool. Redissected by Professor S. G. Shattock, F.R.S. Originally drawn by W. T. Shiells from the specimen (after Irwin Moore).

Shattock refers to the effective closure of the glottis which precedes coughing, and which takes place at the site of the true cords. He states that as the ventricular bands have no muscle, any approximation of them can only be concomitant. However, it seems probable that the ventricular bands can be approximated voluntarily (if the true cords are not approximating normally) by the concerted action of the upper laryngeal muscles, as has been described. Some of the fibres of one or two of these latter muscles are closely associated with the ventricular band.

Howarth (1922) also drew attention to the fact that when coughing or any other spasm occurs the ventricular bands close over the true cords and conceal them from view; but if they are pressed upon with a probe they can be easily displaced, the true cords being revealed in firm apposition. Again, Howarth was referring to the normal larynx, not to the larynx in which there was some interference with the normal function of the true cords by scarring and distortion.

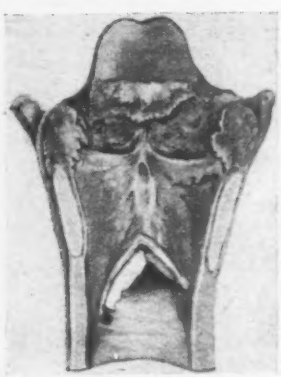

FIGURE XV.

Larynx showing hyperplastic tuberculosis simulating prolapse of the *sacculus ventriculi laryngis*. (Originally drawn by W. T. Shiells from a specimen in the museum of the Hospital for Diseases of the Throat, Golden Square, London; after Irwin Moore.)

In true eversion of the sacculus there may be no symptoms unless eversion of the ventricle follows. If the latter is complete, semi-strangulation of the mass is likely to take place, and a large congested tumour presents in the larynx causing interference with phonation and usually some dyspnœa.

A true eversion of the sacculus appears in the anterior portion of the larynx, but a spurious eversion may do so also. The true cords can be seen to be below and separate from the mass; if there is any doubt, palpation at direct laryngoscopic examination will clarify the position.

The outline of the ventricular band is also clearly separate and above the mass. The mass is capable of partial or complete replacement into the ventricle, but quickly reappears.

XUM

The condition may pass unnoticed for years until a casual or routine examination is made or a sudden congestion is caused by some constriction of the neck of the everted mass with the appearance of symptoms.

If inconvenience is being caused, removal by sharp punch forceps should be adequate in most cases. More elaborate procedures such as laryngofissure may be necessary in very special cases.

Tumours of the Sacculus.

Cystic Tumours.

Cysts of the sacculus are nearly always retention cysts arising in the numerous seromucinous glands of the *tunica propria*. They may or may not cause eversion, and they may become so large as to fill the sacculus and bulge up beside and lateral to the epiglottis, to lie near the base of the tongue in the vallecula of that side, as occurred in Fletcher Ingal's case demonstrated in Chicago in 1899; the size of the cyst may cause the ventricle to evert into the larynx and the cyst itself may bulge into the larynx. In Ingal's case, it bulged upwards to the base of the tongue and into the larynx. On aspiration, it was reduced to the actual everted ventricle (Figure VIII), a flattish tumour five-eighths of an inch by three-eighths of an inch lying above the true cord and below the ventricular band towards the anterior part of the larynx. These cysts, when they just fill the sacculus, have been described as mucoceles of the sacculus, and when infected as pyoceles.

FIGURE XVI.

Drawing of a coronal section of ventricle and sacculus showing hyperplastic tuberculosis and the formation of folds, simulating prolapse, similar to condition shown in Figure XV; the interior of the larynx in this case was also the seat of similar hyperplastic tubercular disease. (Originally drawn from a dissection by Professor S. G. Shattock; after Irwin Moore.)

Solid Tumours.

Solid tumours are as a rule benign neoplasms, and usually fibromata or fibrolipomata. They may cause eversion of either the sacculus or the sacculus and ventricle, a process of turning inside out.

Malignant neoplastic tumours early become fixed by infiltration and would be unlikely to cause an eversion.

Both the cystic and solid tumours are very rare. Reading in 1941 recorded a case of mucocele of the laryngeal ventricle simulating laryngocele.

Summary.

The minute anatomy of the laryngeal ventricle and its appendix, the *sacculus ventriculi laryngis*, is described.

The pathology of the so-called prolapse of the laryngeal ventricle is then discussed. Apparently true eversion seldom if ever occurs, except in association with traction of a cyst or benign tumour, and even then there must be an associated inflammatory factor such as acute or chronic œdema or hyperplasia weakening the attachments of the ventricular walls as well. The negative pressure associated with the coughing which accompanies acute and chronic laryngitis is probably an additional important factor. A condition simulating prolapse of the ventricle occurs in certain pathological states, most commonly in tuberculous infection of the larynx, in which a normal fold in the lower lateral wall of the laryngeal ventricle becomes hypertrophied and projects across the ventricle between the true and false cords. The signs, symptoms and diagnosis and treatment of the true condition are described.

The pathological lesions of the *sacculus ventriculi laryngis* are classified as follows: (i) laryngocele, (ii) hernia of the sacculus, (iii) eversion of the sacculus, (iv) tumours of the sacculus—(*a*) cystic, (*b*) solid.

1. Laryngocele is divided into two groups based on ætiological grounds. Those in the first are atavistic and represent the air sacs which serve the anthropoid apes as supplementary reservoirs for respiratory air, and may be bilateral. In the second group the air sac is the result of a herniation of a portion of the laryngeal wall into the tissues of the neck. This may occur subglottically through the crico-thyroid membrane (contrast tracheoceles through the tracheal wall), and there has usually been a predisposing traumatic or other pathological lesion which has weakened the laryngeal wall. More commonly, it occurs in association with the *sacculus ventriculi laryngis*, which becomes stretched and herniates either up into the vallecula or more often out into the neck. A congenitally long saccule may be a factor in this type. Clinically there are three types: those producing an external sac in the neck and no intralaryngeal signs or symptoms, the wholly internal hernia which extends up to the vallecula and often down into the larynx, and the combined type in which an external neck sac and an internal laryngeal sac are present connected by a narrow pedicle which passes through the thyro-hyoid membrane. The mechanism of the formation of this type is discussed. The signs, symptoms, diagnosis and treatment of laryngocele are then discussed.

2. In hernia of the *sacculus laryngis*, which is akin to the hernial type of laryngocele, increased expiratory pressure causes a medial protrusion of the upper wall of the sacculus at the weak spot between the lower margin of the *aryteno-epiglottideus inferior* muscle and the ventricular band. It may eventually involve the whole of the sacculus except its lowest and outer part. The herniation usually takes place audibly "with a pop", and a tumour the size of a small cherry may appear above the ventricular band, of a paler colour than the remainder of the laryngeal mucosa because of its distension.

Eversion of the sacculus without and with eversion of the ventricle is vary rare by itself, but has been recorded without being associated with a cyst or tumour, when it is less rare. The condition may be simulated by papillary hyperplastic flattened leaf-like processes, which arise from the walls of the laryngeal ventricle in tuberculosis and appear anteriorly between the true and false cords in the position where true eversion of the sacculus appears. The mechanism of production of the true condition is discussed.

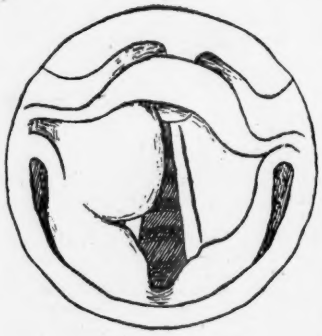

FIGURE XVIIA.

Sketch of the intralaryngeal appearances in the inflated position of Blewett's subject on indirect laryngoscopy. (After Blewett.)

Tumours of the sacculus may be (*a*) cystic, or (*b*) solid. A cystic tumour is usually a cyst arising in one of the seromucinous glands in the *tunica propria*. These tumours have been described as mucoceles, and when infected as pyoceles. Solid tumours are usually benign neoplasms, fibromata or fibrolipomata. They may cause eversion of the sacculus if associated with inflammation. Malignant neoplastic tumours early become fixed by infiltration and are unlikely to cause an eversion.

Reports of Cases.

CASE I.—C.J.B., a male patient, aged twenty-eight years, on September 2, 1948, presented with a swelling in the left side of the neck in front of the sterno-mastoid muscle just below the level of the hyoid bone. He said that it varied in size, and if he squeezed it he experienced an unpleasant taste in his throat. The swelling felt cystic, but because of the unusual story the neck was radiologically examined. No

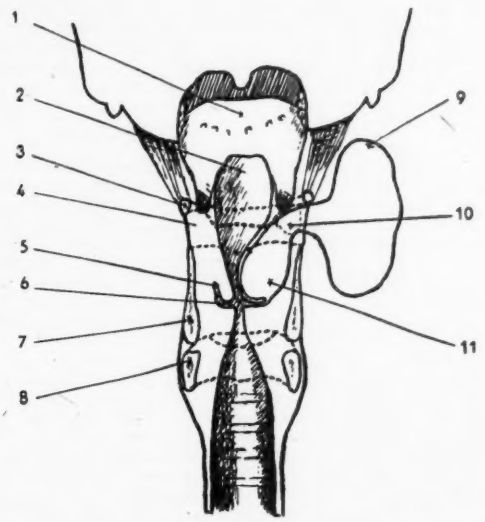

FIGURE XVIIIB.

Sketch by Blewett of the relations of a laryngocele with intralaryngeal and extralaryngeal extensions of the sac, reconstructed from radiographs of one of his subjects as seen from the posterior aspect: (1) base of tongue, (2) epiglottis, (3) greater cornu of hyoid bone, (4) thyro-hyoid membrane, (5) normal saccule or appendix of laryngeal ventricle, (6) ventricle of larynx, (7) lamina of thyroid cartilage, (8) cricoid cartilage, (9) extra-laryngeal expansion of laryngocele, (10) hernial orifice in the thyro-hyoid membrane, (11) intralaryngeal sac of laryngocele.

intralaryngeal abnormality was visible on indirect laryngoscopic examination. The X-ray film showed that there was air apparently above a fluid level in the cyst (see Figure XVIIIA and B). On September 24, 1948, the cyst was removed. At operation the medial end of a fluid-containing,

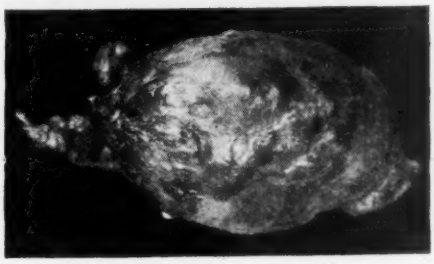

FIGURE XIX.

Case I (C.J.B.): cyst, natural size, removed at operation.

fairly thick-walled cyst tapered off and passed through the thyro-hyoid membrane, in front of the opening through which the internal branch of the superior laryngeal nerve and its accompanying vessels passes, but considerably lateral to the mid-line. It became continuous with the upper part of the laryngeal ventricle, just above which it was ligated and removed. Figure XIX shows the cyst natural size, and Figure XX the low-power and high-power photomicrographic features of its wall.

Although the cyst was only unilateral, because of the above-mentioned macroscopic features and microscopic picture it was judged to fall into the first group described in the text and to correspond to an atavistic remnant of the air sacs found in the anthropoid apes.

CASE II.—D.P., a male patient, aged sixty-three years, presented in May, 1950. (This case is described by courtesy of my colleague, Dr. C. H. Pyman.) For eight years the patient had noticed on coughing a swelling in both sides of the neck adjacent to the larynx, more pronounced on the left side (see Figure XXI). He had first noticed it after a severe bout of hiccups. There was no dysphagia or voice changes and no abnormal intralaryngeal signs were found on indirect laryngoscopic examination. He had a blood pressure of 210 millimetres of mercury, systolic, and 100 millimetres, diastolic. Figure XXII shows the X-ray film. As the condition was not an inconvenience, no treatment was considered necessary, and hence the microscopic structure of the cyst walls was not ascertained.

FIGURE XXI.

Case II (D.P.): photograph showing swelling in left side of neck due to an underlying laryngocele. (By courtesy of Dr. C. H. Pyman.)

The case was considered to be probably one falling in the second group described in the text, the result of herniation of the saccule into the tissues of the neck.

CASE III.—E.E.J. was a male patient, aged forty-five years. (This case is reported by courtesy of my colleague, Dr. C. H. Pyman.) In 1942 the patient had suffered spontaneous hemiplegia while with the army in the Middle East. He developed a cough after this, but recovered from his hemiplegia, except that he suffers from what appear to be epileptic attacks. In 1950 he had a severe paroxysm of coughing and fell on to a fire and burnt himself. It was at that time that attention was drawn to a swelling in his neck on coughing. This swelling was a diffuse one, extending to both sides of the neck at the laryngeal level. Intralaryngeal examination revealed no abnormality. The X-ray appearances are shown in Figure XXIII.

This case was also considered to be one falling in the second group described in the text, the condition being the result of herniation of the saccule into the tissues of the neck.

Acknowledgement.

I wish to thank Mrs. K. Grice for the drawing of Figures I to XVI.

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Legends to Illustrations.

FIGURE XVIIIA.—Case I (C.J.B.): reproduction of X-ray picture (antero-posterior position), showing air in sac over a fluid level.

FIGURE XVIIIB.—Case I (C.J.B.): reproduction of X-ray film (lateral position), showing air in sac and relation to laryngeal framework and hyoid bone.

FIGURE XXA.—Case I (C.J.B.): low-power photomicrograph of cyst wall, showing lymphoid nodes and pseudo-stratified ciliated columnar epithelium (× 200).

FIGURE XXB.—Case I (C.J.B.): high-power photomicrograph of cyst wall (× 400).

FIGURE XXII.—Case II (D.P.): reproduction of X-ray film (antero-posterior position), showing bilateral air-containing sacs in neck. (By courtesy of Dr. C. H. Pyman.)

FIGURE XXIII.—Reproduction of antero-posterior X-ray film in Case III (E.J.), showing air-containing sac clearly on the left side. There is doubt about the shadow on the right side. This X-ray film was taken with semi-inflation. (By courtesy of Dr. C. H. Pyman.)

THE SECRETION OF 3,3′-ETHYLIDENE BIS (4-HYDROXYCOUMARIN) IN MILK.

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THE discovery by K. P. Link and co-workers (1941 and 1943) that certain derivatives of 4-hydroxycoumarin are capable of interfering with the synthesis of prothrombin and other factors essential for blood coagulation has led to the clinical application of these drugs for prophylaxis and treatment of a variety of thrombo-embolic conditions, including those of women in the post-partum period. It has been realized that the "prothrombin" level of patients undergoing dicoumarol treatment has to be controlled continuously in order to avoid catastrophes due to hæmorrhages. In addition to dicoumarol its methyl homologue 3,3′-ethylidene bis (4-hydroxycoumarin) (E.D.C.), has been suggested for practical use (Fantl, 1944; Fantl and Nance, 1947). The latter compound is less potent than dicoumarol. Recovery from E.D.C.-induced hypoprothrombinæmia after discontinuance of the drug is much quicker, a fact indicating greater safety in control (Rose and Krieger, 1951). Another drug of this series, 3,3′-carboxymethylene bis-(4-hydroxycoumarin) ethyl ester, called "Tromexan", has been introduced. The claims for its advantage over dicoumarols are similar to those of E.D.C. (Seegers, 1951). The therapeutically desirable prothrombin level has not yet been established, and it appears likely that other factors beside prothrombin are influenced by the dicoumarol drugs (Hurn and Mann, 1947; Dam and Sondergaard, 1948). Further, an influence on adhesiveness of platelets has been demonstrated by Wright (1945) and a vasodilator effect has been suggested by Gilbert and Nalefski (1948). In this communication we record results of experiments concerned with the transmission of the drug through the milk to the suckling infant.

The technique for the determination of E.D.C. in milk is similar to that suggested by Axelrod *et alii* (1949), and depends on the fact that bis-4-hydroxycoumarin derivatives show in alkaline solution a characteristic ultra-violet absorption spectrum with maximum intensity at 315mμ.

Because of poor recovery of E.D.C. added to animal milk, the original method was modified and the following procedure carried out: to at least three millilitres of milk is added 0·1 millilitre of 2·5N sodium hydroxide solution and the mixture is shaken with ten times its volume of purified petrol ether (boiling point 90°-105° C.) for five minutes. The material is centrifuged and the supernatant fluid is discarded. With the aid of a Pasteur pipette as much as possible from the bottom layer is introduced into a 50-millilitre centrifuge tube (usually 2·6 millilitres are obtained). After addition of 0·25 millilitre of 3N hydrochloric acid and a flake of pepsin, digestion is carried out for two hours at 37° C. A further 0·3 millilitre of 3N hydrochloric acid and water are added to give a final volume of 3·5 millilitres; E.D.C. is extracted by shaking with 20 millilitres of petrol ether for thirty minutes. Fifteen millilitres of the top layer and four millilitres of 2·5N sodium hydroxide solution are shaken for five minutes. The alkaline solution is clarified by centrifuging, and the optical density at 315mμ is determined in a spectrophotometer. A solution of 10γ of E.D.C. in one millilitre of 2·5N sodium hydroxide solution gave an optical density of 0·64. With the above technique, addition of known amounts of E.D.C. to animal milk resulted in 95% recovery.

In order to obtain reliable results, the specimen should contain at least 10γ of E.D.C. As will be seen from the results obtained, the apparent concentration of E.D.C. in the milk samples was too small for identification of the drug. It is therefore undecided whether the observed values are due to E.D.C. or to its metabolic products. In-vitro experiments indicated that treatment of E.D.C.

[1] Part of the expenses of this work were defrayed by a grant from the National Health and Medical Research Council.

solution with N/400 sodium hydroxide solution for five hours at 100° C. resulted in a decrease to 34% of the extinction. From this it would appear that the technique applied has a high degree of specificity.

The patients were treated with E.D.C. within twenty-four hours after childbirth. The last dosage before collection of samples was between 100 and 200 milligrammes of the drug.

Milk specimens as well as blood samples were taken, in the case of humans, approximately twelve hours and, in the case of goats, seven hours after the last dosage. Five guinea pigs forty-eight hours after delivery received a single dose of 30 milligrammes of E.D.C., and specimens were obtained after sixteen hours' isolation from the offspring.

TABLE I.

Subject.	Prothrombin Level. (Percentage of Normal.)	E.D.C. in Plasma. (Milligrammes *per Centum*.)	E.D.C. in Milk. (Milligrammes *per Centum*.)	Amount of Drug Taken. (Grammes.)
Pl. I ..	85	<0·05	<0·03	1·6
Le. ..	48	0·15	0	1·6
Ro. ..	56	0·19	<0·04	1·6
Pl. II ..	15	—	0·15	2·6
H. ..	23	0·18	0·26	1·0
Da. ..	60	—	<0·03	1·2
Goat ..	18	2·2	0·29	1·5
Guinea-pigs (5)	100	—	0·3 (mean)	0·03

With regard to the significance of the figures obtained it should be emphasized that values of the order of 0·1 milligramme *per centum* involve an error of up to 50%.

Discussion.

F. W. Schofield (1924) was one of the first who studied the hæmorrhagic condition in cattle feeding on spoilt sweet clover hay. Link and his team (1941) have shown that the causative agent in this fodder is 3,3'-methylene bis (4-hydroxycoumarin). Schofield noticed that newborn calves appeared to be much more sensitive to the toxic hay than mature animals. J. B. Field (1945) produced a severe reduction of prothrombin levels in baby rats whose mothers were fed with dicoumarol orally. He concluded that the active principle was transmitted through the milk. The concentration of E.D.C. in the milk of humans as shown above is in most cases very low. If we assume human milk production to be approximately 600 millilitres per day, this volume would contain not more than 1·8 milligrammes of the drug.

Objection that metabolic products other than E.D.C. are responsible for the hypoprothrombinæmic effects has been considered, but no experimental evidence for such an assumption has been found. It is very doubtful whether amounts of two milligrammes of E.D.C. given daily to a baby would influence its prothrombin level, even when the physiological hypoprothrombinæmia of the newborn is taken into consideration.

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RENAL TRAUMA.[1]

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ONE'S special interest in a particular subject often starts with being called upon to deal with a number of cases of that condition, and the renal injuries I have seen during recent years form the basis of this paper.

Before the rise of the motor-car and the mechanization of industry, the kidney was a comparatively rare subject of injury. But of latter years the incidence of injury has increased, and seems likely to become even greater. Most reported series of cases in America show a hospital admission rate of about three to four cases per year, but from a checking of the records at the Royal North Shore Hospital of Sydney and the Lewisham Hospital, I would say that in this country the incidence is about half this figure. Owing to the greater exposure to modern hazards and to the greater physical activity undertaken, the injury is much commoner in the male than in the female subject, being in the ratio of about six to one.

It is often remarked that at times even minor trauma can cause a rupture, but I believe that this rarely, if ever, occurs to a normal kidney. In one of my cases the injury was quite trivial, but hæmaturia ensued; this was later shown at operation to be due to a tear through a dilated tuberculous calyx.

A pre-existing renal pathological condition has many times been shown to be a contributing factor. One young man of 15 stone weight seen last year had received a blow in his left loin at football. He had well developed symptoms and signs of a renal rupture, confirmed by retrograde pyelography. At operation I found a large, very hydronephrotic kidney, which had tears through two calyces, one almost severing the lower pole.

These cases emphasize the value of a full investigation, including retrograde pyelography, to which I will refer again later.

It has been stressed also that congenital anomalies of the ribs and vertebræ, by causing an increase in the size of the exposed areas of the kidney, play a part in its susceptibility to injury. I have not been able to confirm this.

Diagnosis.

I do not propose to consider diagnosis in detail, except to note that pain is the chief symptom. Mostly this is more severe in the major injuries, but not invariably so. Macroscopic hæmaturia is a constant sign except in some cases

[1] Read in the author's absence by Dr. Richard G. S. Harris at a meeting of the Urological Society of Australasia on April 18, 1951, at Sydney.

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of mild contusions, or in avulsion of the vascular pedicle. Tenderness and rigidity in the loin are usually present with major ruptures, and a mass also may be palpated. Shock may or may not be present, but if profound, indicates probably severe trauma.

Our biggest problem is, however, not to diagnose a traumatized kidney, but to ascertain the exact extent of its damage. And here, of course, we have two aids—excretion and retrograde pyelography.

Excretion Urography.

I think there can be little argument that in all cases the patient should have an excretion urogram prepared, even if only with a portable machine, as soon as his condition will permit. The uninjured kidney will nearly always be displayed if it is present and has reasonable function, and I believe this to be the main virtue of the procedure. The results seen on the injured side are very variable and may be misleading. Frequently, no dye can be seen at all, and the assumption has been in the past that this is indicative of a more serious injury. However, with the aid of the retrograde method, many patients can be shown to have no break in the kidney substance, and nothing more serious than a contusion. At times a rupture may be present with normal radiographic appearances, or a small tear may allow perirenal extravasation. But it is a safe and readily applicable method, which should be used as soon as the patient has recovered from his initial shock.

Three cases will illustrate this lack of diagnostic certainty.

A patient was injured in the right loin in a motor-car accident, with consequent tenderness in this area and severe hæmaturia. The ninth, tenth and eleventh ribs were fractured on this side. An excretion urogram revealed a normal state on the left side but no excretion on the right. This tended to confirm the diagnosis of right renal rupture of a major degree. However, retrograde studies showed the collecting system to be intact, and in addition indigo carmine excretion was similar from each side. A conservative routine was followed with good results, and I believe that only a contusion or minor tear was present.

The second case was also one of right loin injury. Hæmaturia, tenderness, some guarding and fullness were evident on this side. Normal excretion of dye was seen from the left side, but none from the right. A retrograde pyelogram showed a tear in the upper pole with extravasation, and this was confirmed at operation.

The third patient was a young girl injured while horse riding. When first examined she was suffering from severe shock and moderate hæmaturia, with local bruising pointing to a left loin injury. This was confirmed in the urograms, there being no excretion from this side. A retrograde pyelogram was not made, but a quick cystoscopic examination showed blood coming from the left ureteric orifices. After resuscitation a shattered left kidney was removed.

In all these cases there was no dye excretion from the injured side, and yet the injuries included a contusion, a major rupture and a shattered kidney.

Excretion studies may on the other hand give an excretion sufficient to make a diagnosis of extravasation possible, and they are certainly of great value in drawing attention to a doubtfully injured kidney, and to a hypoplastic or congenital solitary kidney. This latter condition is not excessively rare, but when it is associated with trauma the mortality is high.

Retrograde Pyelography.

Mostly if we are to find out just what happened to the kidney, retrograde pyelograms are essential. Only in this way can we find out with accuracy the site, nature and degree of any rupture, and ascertain in true detail the architecture of the renal collecting system. This is particularly true when pathological changes are already present—for example, hydronephrosis or tumour.

To take pyelograms, cystoscopy is essential, and of course the patient must be fit to undergo the procedure. With cystoscopy unsuspected bleeding bladder lesions have been found, and the indigo carmine test, which I believe to be most useful, can be observed. In addition, blood can be observed emanating from the suspected renal tract.

The value of pyelography has been shown already in the previous cases mentioned, and further examples will confirm it.

After a motor-car accident a man revealed signs and symptoms of a right renal rupture. There were pain and tenderness over this kidney, a mass was palpable and hæmaturia was present. An excretion urogram revealed fairly good excretion from both kidneys, but a soft tissue opacity appeared to cover the lower pole area on the right side. A retrograde pyelogram showed extravasation in this area.

Another patient sustained a blow on the left loin, with resultant shock, pain and tenderness, and the third, fourth and fifth transverse processes were fractured. An excretion urogram revealed a possible extravasation, but a normal outline was seen after retrograde injection. The symptoms were thus considered to be due to extrarenal causes.

After a football match a young man noticed a good deal of blood in his urine, with some pain and tenderness in his left renal area. An excretion urogram showed only a few patches of dye on this side, but at cytoscopy blood was seen coming from the left ureteric orifice, and a retrograde pyelogram with a portable machine showed two large areas of diffuse extravasation. Immediate operation disclosed a kidney with multiple tears, for which nephrectomy had to be performed.

A further patient, after an injury to the left loin, had hæmaturia and pain with muscular guarding. Very little excretion was seen from this kidney with a urogram, but retrograde injection showed extravasation to the medial side of the lower pole. This was not large, so conservative treatment was followed.

I think these cases do show the value of retrograde pyelography, as well as the fact that we can determine with precision the site, nature and degree of any rupture and the extent of extravasation present.

But many do object to the procedure on three counts: (i) the possible introduction of infection, (ii) the possibility of increasing or renewing hæmorrhage, (iii) the view that it is unnecessary if non-operative treatment is preferred. As regards the infection, I do not believe that its introduction is any more likely than after a pyelogram in an uninjured kidney, and there is little evidence in the literature to support the idea. The cystoscopy, like all others, should be performed in an aseptic manner, and the antibiotics are at hand if necessary. It has not occurred in any of my cases.

The danger of increasing the hæmorrhage is also exaggerated, and again the literature does not support this idea. Its seriousness has also diminished with the increasing number of conservative operations now being performed in ruptured kidneys, and by the use of gravity to run the fluid to the kidney for the pyelogram.

The third objection brings us to the treatment of these injuries.

Treatment.

I do not think there is any argument in the consideration of contusions or minor injuries, for which a non-operative routine is followed, or in that of shattered kidneys, for which an operative course is undertaken. But there appear to be two schools of thought regarding the major ruptures. One school strongly advocates no surgical intervention unless there is gross disorganization of the renal pelvis which would eventually lead to a useless kidney. In the great majority of cases the patients do well with rest, sedatives and the prevention of infection. The opposing school believe that not only may lives be saved and renal tissue be conserved, but distal sequelæ may be prevented by conservative operation in all cases in which extravasation is seen. These sequelæ take the form of perinephric abscess, Goldblatt kidney, renal cyst, loss of function, secondary hæmorrhage and hydronephrosis. Many believe that exploration of the area will uncover unsuspected pathological conditions, and that it will also enable a kidney to recover more if its tear is sutured than if it is left to heal amid surrounding blood clot. However, I would repeat that clear renal architecture must be visible by retrograde pyelography if we are to distinguish between parenchymal

tears that do well left alone and shattered kidneys or kidneys with pelvic disorganization that do not.

One patient had sustained a right renal injury two years before which had been treated without operation. During the previous four months she had had repeated attacks of pyelonephritis and cystitis. Urine from the right kidney contained coliform organisms, and the retrograde film revealed a residual cavity above the upper calyx. Other cases of late complications have recently been reported, so that this must be taken into account in a decision whether the kidney should be explored or not.

I am not advocating that all kidneys with a major rupture should be explored, but I do believe that if there are pronounced signs of extravasation, as seen by the results of pyelography, muscle guarding, tenderness and tumour with perhaps increasing pulse rate and signs of infection, then operation is indicated. It should be a conservative operation aimed at saving renal substance by suture, with nephrectomy limited to the shattered or badly torn kidneys. There is said to be less secondary hæmorrhage after suturing than if the kidney is left completely alone, blood clot can be cleared away with less chance of infection arising, and pathological conditions can be dealt with, such as tuberculosis and newgrowth.

There is little necessity for urgent operation. Resuscitation should always be carried out if shock is present, and three days after the injury, when the extravasated blood has coagulated, we can explore almost at leisure. Urine will not be found, as renal secretion is in abeyance for a few days following the injury.

On the other hand, if other severe injuries are present, resuscitation is all that can be done, and will mostly be sufficient to tide the patient over. Conservative treatment will almost certainly carry any patient along who would have withstood operation, and therefore associated abdominal lesions should always be treated first, and alone.

As a summary I would say that with contusions, conservation is best; with shattered kidneys one should operate after carrying out resuscitation; and with major parenchymal ruptures one should operate if the retrograde pyelogram shows well marked extracapsular extravasation.

THE Q–T INTERVAL OF THE ELECTROCARDIOGRAM IN ELDERLY SUBJECTS.

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DURING recent years much attention has been drawn to the *Q-T* interval of the electrocardiogram. Recent evidence that prolonged *Q-T* interval may indicate the presence of serious heart disease (rheumatic fever, myocardial infarction) has provided an impetus to its use in clinical electrocardiography.

In a previous study (Hagen and Freeman, 1950), a series of elderly male subjects was reviewed with the purpose of determining the extent of electrocardiographic evidence of coronary insufficiency in that age group; at that time the *Q-T* interval was unfortunately neglected.

The subjects comprised 52 healthy males between the ages of sixty-five and ninety years. They were all symptomless and quite healthy on physical examination. Their chest X-ray films revealed no cardio-respiratory abnormality and they were free from hypertension. Their ages, *Q-T* interval and cycle length (C) together with Ashman's constant are shown in Table I.

The electrocardiograms taken were those with the three standard leads, also *aVR*, *aVL*, *aVF*, and V_1 through to V_6 inclusive, and in a number of cases *CF2*, *CF4* and *CF6* as well.

A number of formulæ have been devised relating *Q-T* to cycle length (C). The following are the best known: that of Fridericia, $Q\text{-}T = K\sqrt[3]{C}$ where K = 8·22; that of Bazett, $Q\text{-}T = K\sqrt{C}$ where K = 0·37 for men and 0·4 for women; that of Ashman (1942), $Q\text{-}T = K \log[10(C + k)]$ or $K = \frac{Q\text{-}T}{\log[10(C+k)]}$ where C is cycle length in seconds, k is 0·07 and K is 0·377 for men and 0·387 for women (Ashman gives the upper normal limit of K for men as 0·41 and for women as 0·42). As Ashman's formula has been derived from the largest surveys and found most general use it is proposed to evaluate these cases in terms of this formula.

TABLE I.

Case Number.[1]	Age. (Years.)[2]	*Q-T*.	Cycle Length. (Seconds.)	K.[3]
1	68	0·4	0·84	0·42
2	80	0·32	0·72	0·36
3	73	0·36	0·80	0·38
4	76	0·36	0·76	0·39
5	75	0·4	0·96	0·40
6	80	0·36	0·68	0·41
7	73	0·40	1·10	0·38
8	84	0·46	1·12	0·43
9	84	0·32	0·84	0·33
10	74	0·44	1·00	0·43
11	72	0·32	0·88	0·33
12	87	0·36	0·80	0·38
13	78	0·36	0·76	0·39
15	76	0·40	0·96	0·40
16	83	0·39	0·82	0·41
18	76	0·40	0·95	0·40
19	65	0·38	1·00	0·37
20	73	0·36	0·84	0·38
21	77	0·38	1·00	0·37
22	80	0·30	0·60	0·36
23	72	0·35	0·72	0·39
24	70	0·40	0·88	0·41
25	79	0·36	0·80	0·38
26	83	0·40	1·04	0·38
27	72	0·34	0·74	0·37
28	75	0·44	0·68	0·50
29	88	0·36	0·84	0·38
30	81	0·35	0·84	0·37
31	82	0·36	0·80	0·38
32	76	0·32	0·64	0·38
33	78	0·36	0·69	0·41
34	90	0·34	0·82	0·36
35	85	0·34	0·76	0·37
36	71	0·34	0·84	0·36
37	74	0·34	0·76	0·37
38	93	0·36	0·88	0·37
39	76	0·36	1·00	0·35
41	76	0·78	0·50	0·37
43	88	0·36	0·84	0·38
44	67	0·36	0·76	0·39
45	80	0·36	0·80	0·38
46	76	0·36	0·88	0·37
47	73	0·36	0·72	0·40
48	65	0·36	0·80	0·38
49	82	0·34	0·68	0·39
50	80	0·36	0·76	0·39
51	80	0·40	0·96	0·40
52	76	0·39	0·76	0·43
53	77	0·36	0·88	0·37
54	79	0·36	0·80	0·38
55	73	0·43	1·08	0·41
56	78	0·34	0·90	0·35

[1] Total number of cases, 52.

[2] Mean age, 77·5 years.

[3] Mean K, 0·39.

In this series all except six subjects had K values within the limits given by Ashman for younger age groups, and only two of these were far outside the alleged limits of normality.

The average value for K in this age group was 0·39.

This series, although limited, is recorded because hitherto there have been no available data on the normal *Q-T* interval in this age group, in which it is difficult to obtain even a small series of, as far as can be determined, completely healthy subjects, and in the hope that if several similar series are recorded the composite results therefrom may provide some more significant information.

References.

Ashman, R. (1942), "Normal Duration of Q-T Interval", *American Heart Journal*, Volume XXIII, page 522.

Hagen, P., and Freeman, Z. (1950), "The Electrocardiogram in Old Age", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 449.

A FLEXIBLE PLASTIC TUBE ADAPTER FOR HIGH-PRESSURE INJECTIONS.

By DOUGLAS G. RENTON, F.F.A.R.C.S.,
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FOR intravenous injections a flexible tube connexion between syringe and needle is a useful adjunct, particularly if the tubing is transparent. Plastic tubing has this advantage over rubber. The difficulty with these connexions is their aptitude to leak and come apart when pressure is applied. The following is an adaptation of the method of attaching fittings to domestic plastic garden hose.

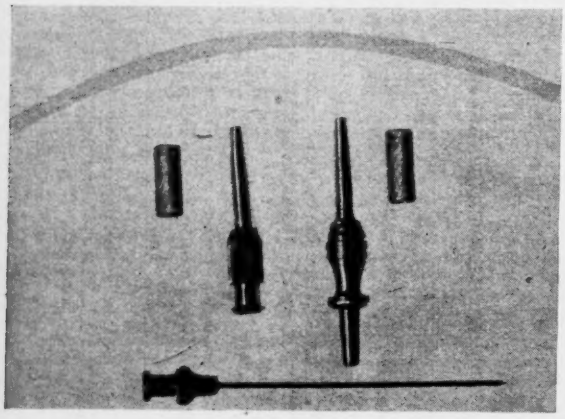

FIGURE I.
The parts.

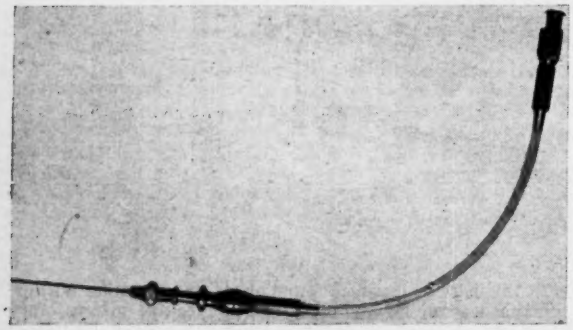

FIGURE II.
The assembled unit.

To a standard male syringe tip or female needle socket is soldered a threaded and drilled shank. The core of the threaded portion is somewhat larger than the outside diameter of the selected plastic tube. After the thread has been made the end is turned down to a taper tip, the smaller diameter of which is equal to the inside diameter of the tube. The retaining cuff is drilled through by a hole equal to the external diameter of the tube. Except for a short section at the end, this hole is enlarged and threaded with a female thread corresponding to that on the shank.

The plastic tube is threaded through the cuff. The end is softened by immersion in hot water, then forced well onto the tapered end of the shank. The cuff is now screwed home securely on the shank. The result is a union which will not come apart when pressure is applied.

This adapter, which is useful for all intravenous injections, was specially made for the rapid injection of opaque medium in angiography and cardioangiography.

Both "Portex" tubing, which will not tolerate autoclaving, and "Nylex" tubing (the commercial plastic insulating tubing), which can be autoclaved, have been used.

After trial of a set of home-made unions, the one illustrated was made by Mr. E. Dixon, the instrument maker to the Alfred Hospital.

Reports of Cases.

LUTEOMA OF THE OVARY.

By RICHARD FLYNN,
Sydney.

M.D., a female patient, aged fifty-one years, consulted me in 1948 on account of generalized hirsutism. She had married when twenty-four years old and was the mother of three children, who were all well. Her menstrual periods had ceased two years previously and she had experienced no inconvenience at that time. She had first noticed the excessive hair as a girl of twenty years. Over the years she had had the hair removed by electrolysis repeatedly without permanent help. In recent years hair had grown more freely on her lips, back and trunk, while on the scalp it did not grow as quickly as formerly.

On examination of the patient's abdomen no abnormal swellings were palpable, and on pelvic examination no abnormal swellings were detected.

Laboratory investigation showed that her blood count was within normal limits and her basal metabolic rate was estimated at +16%.

The late Dr. A. F. Oxenham reported on an excretion urogram as follows:

> Flat skiagrams demonstrating both urinary tracts show the renal areas to be normal in size and situ and free from any shadows of radio opaque density. Urograms taken 5, 10 and 20 minutes after the intravenous injection of Per Abrodil show both kidneys to be excreting the solution satisfactorily and both urinary tracts appear free from any gross abnormality. Lateral skiagram of the pituitary fossa reveals the fossa to be small but its contour is quite regular. There is no suggestion of any evidence of tumour.

Dr. Lee Day estimated the patient's secretion of ketosteroids at 11·7 milligrammes for twenty-four hours. The volume of urine was 1300 millilitres.

Through a low mid-line incision abdominal exploration was carried out on April 5, 1949. The left ovary was adherent to the back of the uterus, and it contained a hard tumour about one centimetre in diameter. The left ovary and Fallopian tube were removed.

The pathologist, Dr. J. M. Garvan, reported as follows:

> The ovary contains a round, well defined encapsulated nodule one and a half centimetres in diameter. It is of light biscuit colour flecked with blood points. Sections of the tumour show it to consist of a sheet of cells with ill-defined outlines but a definite oval nucleus. Lipoid material can be seen as fine droplets. The histology is not clear-cut but the structure resembles that of thecoma or luteoma of the ovary. (See Figure I.)

XUM

Dr. G. Davis concurred in this opinion:

I have seen her on several occasions subsequently and on September 19, 1949, made the following note: "Hair is much less and skin is much clearer"; and again on April 11, 1950: "Hair is again much less and her skin is clearer than any I have seen following removal of suprarenal for hirsutism".

Reviews.

A SYMPOSIUM ON HYPERTENSION.

IN September, 1950, a three-day meeting was held at the University of Minnesota to honour Dr. E. T. Bell, Dr. B. J. Clawson and Dr. G. E. Fahr, who were retiring from their chairs in the Faculty of Medicine after long and distinguished service. The meeting took the form of a symposium on hypertension and a number of original workers in this field were invited to contribute. The present volume records the proceedings of the meeting.[1] The 30 papers form a convenient summary of present knowledge of the subject from the experimental, clinical and pathological aspects. Bell and Clawson each contribute papers on the pathological anatomy of hypertension and Fahr writes of hypertension in chronic genuine nephrosis. On the experimental side Goldblatt, Grollman, Pickering, Page, Shorr, Braun-Menendez and Selye, through their papers, summarize very well most of the accumulated experience of the last seventeen years. Chasis has some second thoughts on the renal hæmodynamics of human essential hypertension and there are several papers dealing with the adrenal cortex. Wilkins, Smithwick, Nickerson, Chapman, Wolff, Page, Taylor and Corcoran deal adequately with therapy. Hypertension has become such an enormous problem that it is becoming extremely difficult even for investigators in this field to keep completely informed on current work. To them and especially to the clinician who has not had time to consult the earlier papers this book will prove useful. There is very little that is new, but it is clear that most contributors have taken considerable trouble to marshal the facts and to look for the signposts that point to future advances. The book is well produced and certainly should be available in every medical library.

A TEXT-BOOK OF PATHOLOGY.

TEN years elapsed between the appearance of the Fifth Edition of Muir's Pathology and the Sixth Edition, which is now available.[2]

This text-book established a high reputation with its earlier editions, a reputation well maintained in the latest edition, the revision of which is by Professor D. F. Cappell. The original character of the book has not been changed, nor has the mode of presentation been varied, but considerable new information is added and this has resulted in the addition of a hundred pages to the volume.

The paper is an improvement on that of the preceding edition and this accounts for the sharper definition of the illustrations and for the greater clarity of the text.

It is a difficult task for a young pathologist to revise a text-book written by his former chief. Professor Cappell is to be commended highly for the judgement he has shown in blending the new, his own additions, with the old, the work of Sir Robert Muir.

This volume will do much to maintain and indeed enhance the reputation Scottish schools have long held for sound knowledge of pathology. So much new information appears in this book that it is difficult to choose outstanding additions, but perhaps special attention should be drawn to the hæmopoietic system, in particular to the classification of the primitive red cell precursors, and also to the respiratory system, in particular to coal-miner's lung and to bronchial carcinoma.

The description of intestinal parasites in the earlier editions has been omitted from this one. A brief bibliography is introduced at the end of the text.

To sum up, author and publishers alike are to be congratulated on the production of this volume, which is sure to remain a standard text-book of pathology.

EXPERIMENT IN DENTAL CARE.

THE author of "Experiment in Dental Care", J. T. Fulton, D.D.S., by virtue of a fellowship grant from the World Health Organization, a Department of the United Nations, has joined those overseas authorities who have made a personal examination of New Zealand's use of school dental nurses to provide State dental treatment for children, together with the country's more recent efforts to provide dental care for adolescents.

Dr. Fulton's monograph[1] is an excellent and comprehensive analysis of the incidence of dental caries in the New Zealand child population, and of the objects of and the degree of success attending the efforts of the Government of that country in dealing with the problem. It also outlines the history and organization of the scheme, and gives an analysis of its costs and administration. Further chapters are devoted to the training of the dental nurses operating in the organization and the circumstances surrounding the dental profession in New Zealand.

The material is very well collated and presented, and amply illustrated by carefully prepared graphs.

The author, in his approach to the experiment which, as he states, "has been a controversial subject among dentists the world over", has set out to provide from his own extensive observations certain facts which, however, do not extend beyond the field of caries control by reparative and dental health educational measures which constitute the scheme. He records the experiment's success within this limited scope.

Advisedly, in our opinion, he points out the limits of his report and adds that no attempt has been made to evaluate the programme in terms of what is ideal care for the child or to consider other aspects of dental health of the child, such as the health of the supporting tissues and the extent of malocclusion. As this booklet is published for wide distribution, it appears to us that, in addition to his observations outlined above, the author might have further stressed the fact that the experiment is obviously a partial, though laudable, approach to the overall problem of the dental care of children, in that the experiment requires, to become a complete approach, parallel schemes of other types of treatment, prevention and research. This would have overcome the tendency of the lay reader to regard this experiment as the complete answer to the problem of dental care for children.

A PATHOLOGIST REVIEWS HIS PAST.

IN a pleasantly interesting book, "Leaves from a Doctor's Life", Sir Philip Panton, a distinguished London pathologist with a distinct flair for elegance of literary style, gives a witty and rather puckish account of his impressions and experiences after reaching the allotted span in a most eventful life.[2] With a whimsical sense of humour he writes entertainingly about his school days; and here is the type of incident that comes readily to his nimble mind: "A few years later, my housemaster made a correction in one of my Latin verses; but I could see nothing wrong with my effort; nor could I read his correction; so I took the thing back to him, and said I was sorry but I couldn't understand what was wrong. The master looked at the correction for a long time and at all angles, finally bringing a magnifying glass to bear upon it. At length he deciphered the words and read out a little sheepishly: 'Write better.' Fortunately, he did not suspect me of guile, and indeed I had none."

Then again, as an old Harrovian Sir Philip seems to have some kindly recollections of the headmaster, Dr. Welldon,

[1] "Hypertension: A Symposium", edited by E. T. Bell, M.D.; 1951. London: Geoffrey Cumberlege, Oxford University Press. Melbourne: Oxford University Press. 9½" × 6¼", pp. 584, with 121 illustrations and figures. Price: 78s. 9d.

[2] "Muir's Text-Book of Pathology", revised by D. F. Cappell, M.D., F.R.F.P.S.G.; Sixth Edition; 1951. London: Edward Arnold and Company. 9" × 6", pp. 1110, with 636 illustrations. Price: 50s.

[1] "Experiment in Dental Care: Results of New Zealand's Use of School Dental Nurses", by John T. Fulton, D.D.S.; 1951. Geneva: World Health Organization. 9¼" × 6¼", pp. 88, with 12 illustrations. Price: 5s.

[2] "Leaves from a Doctor's Life", by Philip Panton; 1951. London: William Heinemann (Medical Books), Limited. 8½" × 6", pp. 240, with nine illustrations. Price: 22s. 6d.

and recalls an incident connected with the religious instruction regularly provided at his old school: "There was to be seen about the High Street in those days a large, slovenly, pot-bellied gentleman, usually the worse for beer and known to us as 'Old Bottles'. The name was a periodic source of discreet merriment in chapel when Weldon boomed out the passage about putting new wine in old bottles."

And so the story goes on. He tells about his days as an undergraduate at Trinity College, Cambridge, of his early professional experiences, and of his associations with many important personages and institutions throughout his career as a specialist. One amusing anecdote is related about the great neurologist Sir Henry Head, whose weakness in one respect made him slow to admit his ignorance of any subject: "Once Wm. Bulloch, the bacteriologist, played a rather unkind trick. He asked Head at luncheon in the hospital dining-room if he had read Hagenheimer's new book on locomotor ataxia. And Head replied that so far he had not had time to do more than glance through it. 'Well, you have done more than the rest of us', said Bulloch. 'There is no such book.'"

Readers who are curious to know what a pathologist thinks of hospitals, medical students, examinations, degrees, patients, and the doctors who attend them, are advised to spend a few hours alone with Sir Philip Panton.

A YEAR BOOK OF GENERAL SURGERY AND ANÆSTHESIA.

THE material included in "The 1951 Year Book of Surgery"[1] is drawn from journals received by the editors between July, 1950, and May, 1951. It covers a wide range of general surgery as well as anæsthesia, which has a special section. The current outlook of surgery is reflected in the early part of the volume. After a short chapter on general surgical topics, burns and shock, fluid and electrolyte balance, and nutrition are dealt with. The circulatory aspects of burns and shock occupy most of this chapter; two abstracts refer to ACTH and cortisone. The role of potassium and potassium deficiency are prominent in the chapter on fluid and electrolyte balance. Then follow chapters on wounds and infection, and on antibiotics, chemotherapy and antiseptics. The chapter on neoplasm is short, but the subject is prominent throughout the rest of the volume, notably in the chapters on the thyroid, the breast, the lungs and thoracic cavity and the various parts of the alimentary system. The surgical management of congenital abnormalities dominates the chapter on the heart and major blood vessels. Sympathectomy has a chapter to itself, as do hernia, the adrenal gland and surgical armamentarium. The remaining material is grouped on an anatomical basis. Special note may be made of reports on the surgical treatment of pulmonary tuberculosis, the technique of surgery of the biliary tract and of the portal venous system, and liver function in relation to surgical operation.

The section on anæsthesia occupies nearly 80 pages. It has chapters on agents and techniques for special situations (particular operations and certain groups of patients), physiological changes during anæsthesia, drugs for local analgesia, spinal analgesia, analgesic drugs, intravenously administered barbiturates, muscle relaxants, inhalation therapy and endotracheal technique.

In both sections editorial comment is rare and brief. The excellent general production, handsome binding and wide range of material abstracted make up a volume that will be useful to all concerned with surgery and anæsthesia.

A YEAR BOOK OF MEDICINE.

LIKE its immediate predecessor of 1950, "The 1951 Year Book of Medicine"[2] is divided into five parts, each with its own editor. Paul B. Beeson deals with infections, J. Burns Amberson with the chest, William B. Castle with the blood and blood-forming organs, Tinsley R. Harrison with the heart and blood vessels and the kidney, and George B. Eusterman with the digestive system. The volume is made up entirely of abstracts from journals received by the editors between May, 1950, and May, 1951. Critically but briefly annotated by the editors, these abstracts should provide the reader with most of the significant material in current essentially medical literature. Part I, which deals with infections, opens with sections on chemotherapy of infection and ACTH and cortisone in infection, followed by some thirty specific forms of infection, and closes with sections on the collagen diseases and on theoretical aspects of the subject, including a long abstract of F. M. Burnet's paper on the general pathology of virus infections. In Part II the chest is considered in sections on physiological mechanisms, diagnostic and therapeutic procedures, pneumonia, tuberculosis, mycoses, bronchiectasis, bronchial asthma, emphysema, pnemonokoniosis, sarcoidosis, neoplasms, miscellaneous and unusual pulmonary diseases, and diseases of the pleura. The arrangement in Part III is the same as in the previous volume and follows a conventional grouping into the main clinical disorders associated with the blood and blood-forming organs. Part IV covers the heart and blood vessels and the kidney, with sections on diagnosis, congenital heart disease, rheumatic fever and bacterial endocarditis, hypertension, impairment of the coronary circulation, arteriosclerosis, congestive heart failure, arrhythmias, electrocardiographic and other procedures, peripheral vascular disease, thrombo-embolic disease, cerebral vascular disease, miscellaneous subjects and the kidney. Part V is in four sections according to a simple anatomical subdivision of the digestive system. The improvements in the Year Book series adopted last year, in the way of increased size of the volume and attractive binding, are maintained.

[1] "The 1951 Year Book of General Surgery (July, 1950-May, 1951)", edited by Evarts A. Graham, A.B., M.D., with a section on anæsthesia edited by Stuart C. Cullen, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 8" x 5¾", pp. 630, with 209 illustrations. Price: $5.00.

[2] "The 1951 Year Book of Medicine (May, 1950-May, 1951)", edited by Paul B. Beeson, M.D., *et alii*; 1951. Chicago: The Year Book Publishers, Incorporated. 8" x 5¾", pp. 696, with 149 illustrations. Price: $5.00.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"The Use of Tracer Elements in Biology", by W. G. Overend, Ph.D.; 1951. London: William Heinemann (Medical Books), Limited. 7½" x 5", pp. 62. Price: 3s. 6d.

A monograph outlining the applications of isotopic and radioactive tracers to the study of biological problems.

"Liver Injury: Transactions of the Ninth Conference, April 27 and 28, 1950, New York", edited by F. W. Hoffbauer, M.D.; 1951. New York: Josiah Macy Junior Foundation. 9" x 6¼", pp. 232, with 93 illustrations, eight in colour. Price: $3.00.

Six subjects are dealt with by papers and discussion.

"Renal Function: Transactions of the Second Conference, October 19-20, 1950, New York", edited by Stanley E. Bradley; 1951. New York: Josiah Macy Junior Foundation. 9½" x 6¾", pp. 178, with 31 text figures. Price: $3.00.

Seven subjects are dealt with by papers and discussion.

"The 1951 Year Book of Radiology (June, 1950-June, 1951)"; Radiologic Diagnosis edited by Fred Jenner Hodges, M.D., and John Floyd Holt, M.D.; Radiation Therapy edited by Harold W. Jacox, M.D., and Vincent P. Collins, M.D.; 1951. Chicago: The Year Book Publishers, Incorporated. 9¾" x 6¾", pp. 394, with 368 illustrations. Price: $7.00.

One of the "Practical Medicine Series" of year books.

"Studies from the Institute for Medical Research Federation of Malaya", by various authors; 1951. Jubilee Volume No. 25. The Institute for Medical Research, 1900-1950. Kuala Lumpur: The Government Press. 10" x 7½", pp. 404, with 112 illustrations.

Deals with the development of the Institute of Medical Research and with historical trends in Malayan medicine.

"Clinical Pathology of the Eye: A Practical Treatise of Histopathology", by Bernard Samuels, M.D., and Adalbert Fuchs, M.D.; 1952. New York: Paul B. Hoeber, Incorporated. 11" x 8", pp. 438, with 418 illustrations, 191 in colour.

Intended for the pupil and to be used by the teacher as a basis of instruction and also for the ophthalmologist and the general practitioner and surgeon.

The Medical Journal of Australia

SATURDAY, MARCH 22, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

UNIVERSITY DEVELOPMENT IN AUSTRALIA.

ABOUT three years ago a discussion took place in these columns on universities and their problems. Reference was made to an investigation by Nuffield College into "The Problem Facing British Universities". It was shown that the demand on universities today comes from many quarters and is greater than it was before the last war. It was also shown that the demand for university education is not the same in all countries. There are many possible reasons for this and the reasons include the system and grading of schools, the social organization and general attitude of people, and also what we called community habits. The proportion of the population which enters universities in Scotland is twice as large as in England. Again, compared with Great Britain, the United States with three times the population had in 1940 thirty times the number of full-time college and university students. In Australia the numbers of university students have increased enormously since the end of the last war.

Reference to the subject of Australian universities has been prompted on this occasion by the appearance in the *University of Melbourne Gazette* of an article entitled "University Development in Australia" by Sir Douglas Copland, the Vice-Chancellor of the National University in Canberra. Sir Douglas Copland makes the bald statement that as a nation Australia has neglected her universities. Since 1945, he thinks, the university development in this country has not kept pace with the requirements of a progressive and expanding economy. He points out that Australia emerged from the war with six State universities and two university colleges, all of which had built up imposing academic records. By and large, however, "their work was confined to undergraduate teaching; there were important gaps in their work, in particular in post-graduate work and research activities; and some important studies, e.g., those concerned with the languages and culture of our neighbours to the north, were very much neglected". After the war, enrolments, which in 1939 had been only 14,000, rose quickly to 32,000 with the great influx of returned servicemen students. Running costs of the universities rose from £850,000 in 1939 to £3,500,000 in 1950. Fees and endowment income did not rise proportionately, and although State Governments increased their grants from £350,000 to £1,250,000, the position of the universities would have been very serious if the Commonwealth Government had not become an important contributor to university finances. Readers will not need to be told that the costs of the universities have risen still higher since 1950. Sir Douglas Copland tells us that early in 1950 the Commonwealth Government appointed a committee of inquiry into the needs of universities and that as a result of the interim recommendations made by the committee the Commonwealth Government is providing the States with from £800,000 to £1,000,000 *per annum* for the next three years, the amount depending on the level of the States' own grants to their universities. The new Commonwealth grants will replace the research grants and the Commonwealth Reconstruction Training Scheme subsidies previously paid by the Commonwealth Government. While this must give a certain amount of satisfaction, it is pointed out that about one-third or more of the new money will be needed to cover what would otherwise have been deficits, but most of the rest will be absorbed in cost increases and higher salaries. In other words, little money will be available for new developments. In university matters, as in so many other public activities, we seem to be destined always to pursue and never to overtake. The numbers of students are certain to increase and Sir Douglas Copland reminds us that under the Commonwealth scholarship scheme every good student who is capable of undertaking a university course will be provided with financial assistance and the opportunity to enrol as a student. Sir Douglas Copland sets out the estimated numbers of university enrolments in future years, and concludes that in another ten years we shall have nearly three times the number of university students that we had in 1939. If we exclude the National University at Canberra and the New South Wales University of Technology, only the same university structure, however, will be available in ten years time as we had in 1939. The numbers of students at the universities of Sydney and Melbourne are in each instance more than twice as great as the numbers attending the two largest English universities, those of Manchester and Birmingham. Basing his statement on the belief that the optimum size of a university is 4000 students, Sir Douglas Copland states that there should be blueprints for at least another two university institutions in each of the States of Victoria and New South Wales and another one in South Australia and in Queensland. These institutions should take the form of university colleges linked for the time being to the existing universities. Two large gaps in the Australian university structure have, in the opinion of Sir Douglas Copland, been post-graduate training and research on the one hand and the teaching of applied technology on the other. He points out that to some extent these gaps are being filled by the establishment of the Australian National University at Canberra and the New South Wales University of Technology. We need not follow Sir Douglas Copland in his further comments or in his estimate of university expenditure which is needed in Australia.

In the two last sentences of his article Sir Douglas Copland states that in terms of bread and butter, or in the more abstract terms of cultural development, a programme of university expansion will pay handsome dividends. He also states that the defence and economic prosperity of Australia, no less than scientific and cultural development, depend upon the maintenance of a high standard of living in the universities. Both these statements are undoubtedly true; they are likely to appeal to citizens who always look for a *quid pro quo* in any public or semi-public undertaking. In discussions of this kind, some reference should be made to the general purposes of a university and of university education. It is commonly held that the purpose of a university is to teach; this is one of its purposes, but by no means the most important. A university is a place where students can undertake certain courses of study, so that after satisfying the examiners they will be able to take their places in society and earn their livings in the subjects of their choice. To look at a university from this point of view only is not enough. We may be quite certain that if an attempt was made to confine university activities to these utilitarian ends the institution would deteriorate and eventually lose all real value. A university must give its students something more than technical knowledge. It should produce educated men and women, or at least men and women who know what education means and have realized the value of its pursuit throughout life. In the Nuffield College report to which reference has been made, we read that "there is always an element of general education or culture in the teaching a university gives; . . . this teaching is given by scholars and scientists who are themselves working at the frontiers of knowledge". This statement points to the real purpose of a university, which is its existence as a centre of thought and the pursuit of knowledge. This should be much more emphasized than it is today. Thought is impossible without a certain amount of freedom from routine duties. Universities should have on their staffs persons who are capable of constructive thought, for they have to influence the minds of their students and teach them how to think. These staff members have also to act as a thought centre from which the whole community —its strata and all their members—will derive benefit. They are bound to fail in this if they are overburdened with routine duties. In all urgently needed propaganda for the development of Australian universities, increasing emphasis should be laid on the real purposes of universities and university education. Every member of our democratic community can be made, and should be made, to realize this.

Current Comment.

ABUSE OF THE BARBITURATES.

In this country, as elsewhere, the barbiturates are prescribed freely, even though within the provisions of control legislation, and the common medical opinion is probably that much good results from this practice, with little if any harm. No doubt many practitioners can say that they have had no first-hand experience among their patients of addiction, poisoning or other abuse, and they wonder what all the fuss is about—for there is a degree of fuss, as is evidenced, for example, by the persistent recurrence of articles in medical journals on some aspect of the subject. There seems no reasonable ground for doubt that the barbiturates are habit-forming drugs; this has been well demonstrated by the work of Isbell and his co-workers, to which we referred in a leading article on June 23, 1951. Moreover, even if addiction to the barbiturates is acquired much less readily than that to, for example, morphine, Isbell's evidence suggests that its manifestations are in most ways much more serious than those of addiction to morphine. For these reasons and because of, rather than despite, the absence of the dramatic from the common experience of prescribers of barbiturates, a constant reminder of their potential dangers is important. These dangers and their insidious nature are brought out by J. M. Maas[1] no less strongly because he inclines to the view that habituation rather than true addiction follows the use of large doses of barbiturates over a long period. One important point he makes is that while it is admittedly true that innocently acquired habituation to the barbiturates does exist in some cases, there is ample evidence that in many instances the habit is brought about by a number of extraneous factors. These include (he writes of the United States, but the facts can, no doubt, be more widely applied) improper methods of distribution of these drugs, the search on the part of individuals to find an escape mechanism from realities, and the use of these agents to compensate for the accelerated pace of living so prevalent today. In other words, it should not be thought that all cases of habituation are iatrogenic, or that the iatrogenic factor, when present, is always the most important. Moreover, the sound physician will exercise sufficient supervision of patients receiving sedative drugs like the barbiturates to ensure that even the "small minority" (Maas's words) who develop mild habituation are suitably handled. Unfortunately, Maas points out, there appear to be overworked, careless or incompetent doctors who prescribe sedatives instead of seeking a specific diagnosis with appropriate treatment, and an irresponsible minority (with whom the law eventually catches up) who will prostitute their profession for financial gain. From first-hand knowledge of United States conditions Maas states that the worst offenders are drug peddlers—rare unscrupulous pharmacists (Maas stresses their rarity), night club waiters and unauthorized visitors to high school and college campuses. Apparently one popular and extremely dangerous form of abuse is the concomitant use of alcohol and barbiturates to produce brief intense exhilaration followed by profound intoxication and narcosis; this frequently causes death, even with non-toxic quantities of each drug. Another practice, which is, as Maas states, even more vicious and insidious, consists in the ingestion of cerebral stimulants such as the amphetamine compounds upon arising and during the day, followed by heavy barbiturate medication at the time of retirement. This practice, which is said to be common amongst "desk" workers under constant intense tension and pressure, entertainers and chronic devotees to night life, is aimed at extracting more out of its deluded victim's wakeful and sleeping hours than is physiologically normal. It sets up an abnormal cycle which can be difficult to correct, and the final results may be disastrous.

Treatment and rehabilitation of the barbiturate habitué are often very difficult, particularly as many, like a large group of the chronic alcoholics, do not really wish to break the habit; to do so brings the intolerable task of facing reality. In any case treatment is a task for the expert. Methods of prevention should concern everybody who has the legal right to prescribe barbiturates, or indeed who has any sense of responsibility in a community. Maas's considered opinion is that a panacea can never be hoped for, but much can be done. Even the best-framed legislation is not free from fallacy and, as Maas points out, will probably not materially reduce the quantity of barbiturates legitimately prescribed and employed. Nevertheless, it warrants the most conscientious cooperation of every practitioner. Some have suggested, and one ethical pharmaceutical house supplies, a combination product

[1] *The American Journal of the Medical Sciences*, August, 1951.

containing minute quantities of an emetic drug with barbiturates. The theory is that ingestion of excess quantities of the product causes expulsion of the hypnotic by vomiting, but in practice the idea has serious defects and even dangers. The practitioner's greatest obligation is to exercise care and intelligence in the prescription of barbiturates after a conscientious evaluation of the patient's condition. It is important not to be stampeded by complaints of simple insomnia; simpler measures than the taking of drugs are often sufficient, and it is well to bear in mind that the patient who loses sleep tends to make it up at other times. Attempts should be made to establish a normal rest pattern in each individual case and to avoid the abnormal patterns which can become established insidiously with the taking of barbiturates at odd hours. Another essential point is cooperation between medical practitioners and pharmacists. The problems associated with the abuse of such widely used drugs as the barbiturates are necessarily much more subtle than those of drugs like morphine, heroin and cocaine, and constant vigilance is called for. Incidentally it seems reasonable to comment, though Maas does not mention the point, that the uncontrolled distribution of amphetamine compounds, apart from other disadvantages, cannot be expected to decrease the illegitimate demand for barbiturates.

RENAL REGULATION OF THE EXTRACELLULAR FLUID.

THE old description of the function of the kidney was that this organ eliminates unwanted products of metabolism, or simply waste matter. When attention was directed to the importance of metallic balance in physiological saline solution and therefore in blood, a new attitude arose which emphasized metallic ratios. Diabetes in particular introduced the conception of threshold. The problem which perplexed Barcroft, "How can it regulate the blood concentration of so many substances simultaneously?", prompted Cushny to put forward his hypothesis that the glomerulus acts as a jelly filter (Starling) whilst the tubules absorb a standardized Locke solution. A notable contribution to renal physiology which emanates from the department of physiology in the University of Melbourne proves clearly that the action of the tubules is more complicated than the Cushny hypothesis indicates.[1] Briefly the main findings of this important work are as follows. When sodium is abstracted from the body the kidney cannot find sodium replacement or produce a cation to act as a substitute, but eliminates chlorine so as to restore the old ratio, though the osmotic pressure may be disturbed. On the other hand, if chlorine ions are abstracted there is not an elimination of sodium to maintain the balance, but the anion HCO_3 takes the place of the chlorine. The kidney, which regulates every ingredient of the blood, and therefore of the extracellular fluids, except protein and carbon dioxide, is exceedingly sensitive to any departure from the electrolyte pattern in the glomerular filtrate. The technique of producing distortion of this pattern by abstraction of one ingredient is new and opens up a wide field of investigation. The inquiry is by no means academic, for distortion of the electrolyte balance can readily occur through loss of pancreatic juice (duodenal fistula) in which the blood sodium level is lowered, through burns, excessive sweating, persistent vomiting (in which chlorine loss may be considerable), and other conditions. As the authors put it, the constancy of the chemical pattern of the blood, and therefore of the extracellular fluids, "suggests that this pattern reflects a very basic genetic character of animal life".

A concept arising from these findings is that the administration of physiological saline may actually do harm when it does not counterbalance a departure from the normal ratios; logically saline should be compounded to correct the blood conditions in the patient just as, in the physical sphere, astigmatism is corrected by a lens with adjusted and calculated radii of curvature. One difficulty encountered by the investigators concerns acidosis, for they find that the behaviour of the kidney in response to acid reaction following abstraction of sodium is very different from the renal response when the acidity arises from organic acids. The authors suggest that here the difference may be the outcome of the kidney's efforts to preserve the balance between sodium and chlorine ions. Though calcium and potassium abstraction are discussed, the main argument centres around the balance between sodium and chlorine. A very interesting criticism is given of the concept of threshold, which the authors are inclined to challenge. One cause of the apparent threshold may simply be that the mechanisms of specific absorption have been given more work than they can accomplish—the apparatus becomes clogged, so to speak, and so excess passes into the urine.

Though the exposition is in places unduly wordy, this contribution to the physiology of the kidney tubules is of major importance and the authors and the school from which their work emanates are to be congratulated. There is a voluminous bibliography, but the name of E. H. Starling is missing.

[1] *Acta medica Scandinavica*, 1951, Supplementum 261, "Renal Regulation of the Extracellular Fluid II, Renal Physiology in Electrolyte Subtraction", by D. A. Denton, Victor Wynn, I. R. McDonald and Shirley Simon, Melbourne.

INTRAVENOUS IRON THERAPY.

IRON SALTS given by mouth are often poorly tolerated, and absorption may be incomplete. In some cases of anæmia the patients do not react at all to orally administered iron. For many years attempts have been made to find an iron compound which could be administered intravenously without producing toxic symptoms. Such substances as ferric hydroxide, ferric oxide and ferrous ascorbate produced maximum reticulocyte response, but the toxic symptoms were severe enough to preclude their use for therapeutic purposes except under most unusual circumstances. These substances apparently flocculate the blood on contact, the resulting emboli moving to the lungs. It has been noted that injections of saccharated iron into mice do not bring about the deposit of iron in the lungs. Several clinical investigations with intravenous injections of saccharated iron have been made. The latest and most complete investigation has been made by R. G. Holly.[1] In all 23 patients were studied; 11 were suffering from iron-deficiency anæmia with a hæmoglobin value below 10·5 grammes *per centum*; five had pregnancies normal by usual obstetric standards with a hæmoglobin value above 10·5 grammes *per centum* but lower than the normal value of 13·4 grammes *per centum* for the non-pregnant female; three had post-partum anæmia; four had gynæcological conditions with anæmia. The hæmatological response was good in 21 cases; in two cases continued bleeding prevented good response. Toxic symptoms were very slight; only one patient had a generalized reaction with epigastric pain, faintness and a throbbing headache, and this passed off within five minutes. Total doses of saccharated iron ranged from 480 milligrammes to 1430 milligrammes, but a 500 milligramme total dose has been found to be quite effective in three single daily doses of 100 milligrammes or five millilitres, 200 milligrammes or ten millilitres, and 200 milligrammes or ten millilitres. The injections were given slowly with a fine needle. Saccharated iron is a colloidal solution of iron hydroxide stabilized with alkali and cane sugar containing 20 milligrammes of iron per millilitre. The hæmatological responses were immediate, the hæmoglobin increasing steadily for the period of observation up to the capacity of the marrow. The toxicity of preparations for intravenous use is apparently related to the degree of ionization of the iron in the compound. Sucrose forms with iron a complex in which the degree of ionization is not great. The iron is probably removed from the circulation by the reticulo-endothelial system and dissociates at a rate not toxic to the body. Iron not used directly for hæmoglobin synthesis is stored.

[1] *Blood: The Journal of Hematology*, November, 1951.

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Abstracts from Medical Literature.

OPHTHALMOLOGY.

Diathermic Coagulation for Retinoblastoma.

Charles A. Perera (*American Journal of Ophthalmology*, September, 1951) reports a case of bilateral retinoblastoma, in which diagnosis was established by enucleation of one eye and diathermic coagulation of the tumour mass was carried out in the other eye. The mass was situated in the upper nasal quadrant between the equator and the *ora serrata*. Radiation was considered unsuitable, as such treatment to a mass so anterior was likely to damage the eye and render it useless. The growth was outlined by a line of diathermic applications and the area within this space was then thoroughly treated; a 1·5 millimetre penetrating electrode was used, each application lasting three seconds. Two years later the vision was normal, and there was no evidence of recurrence.

Galactosæmia Cataract.

Harold F. Falls *et alii* (*American Journal of Ophthalmology*, September, 1951) report a case of galactosæmia and review the literature. They state that examination of the patient showed a sharply outlined zonular area of increased density of an opalescent nebular nature just beneath the capsule in both lenses. The nuclear areas were also somewhat opalescent in character. The authors point out that in the early stages the lens changes are reversible, and for this reason it is essential to make the diagnosis and initiate treatment before permanent tissue changes have occurred.

James H. Enns (*American Journal of Ophthalmology*, September, 1951) reports a case of galactosæmia cataract. He states that galactosæmia is a congenital anomaly usually making its appearance within the first few months of life and is characterized by hepatomegaly, galactosuria and galactosæmia with impaired lactose tolerance, loss of and failure to gain weight, lethargy, mental dulness, absence of interest in surroundings, cataracts, albuminuria without impairment of renal function and secondary anæmia. The disease is due to inability of the liver to convert lactose to glucose. In the case reported there was a moderately dense homogeneous relucency entirely throughout the fœtal and embryonal nuclei with a few scattered small brilliant white opacities. On a galactose-free diet clearing of the nuclear haze and disappearance of the cortical opacities occurred.

Rose Bengal Test in Diagnosis of Deficient Tear-Formation.

H. Walter Forster (*Archives of Ophthalmology*, April, 1951) examines the rose bengal test and compares it with the Schirmer test in the diagnosis of *keratoconjunctivitis sicca*. He states that rose bengal may be used in the conjunctival sac in a concentration of 1%. The instillation is uncomfortable, but the irritation wears off after irrigation or instillation of pantocaine. The local anæsthetic may be instilled first and so irritation avoided. In eyes with considerable deficiency of tear-formation, rose bengal stains the bulbar conjunctiva and caruncle intensely in the exposed area between the lids. In cases of corneal involvement the epithelial filaments also take the rose stain, whereas the areas of Bowman's membrane from which the filaments of epithelium have come loose do not. The author compares the rose bengal test with the Schirmer test in 98 cases. He states that there was good correlation between the Schirmer and the rose bengal test in the cases of definitely deficient tear-formation. Thirty of the eyes yielded mildly positive results from rose bengal tests, and for 23 of them the results of Schirmer tests were negative. It seems that the result of the rose bengal test is more likely to be definitely positive than that of the Schirmer test in early or mild cases. There is no doubt that deficient tear-formation will produce devitalized conjunctival epithelium, which takes the rose bengal stain. The staining reaction with rose bengal is not apparently affected by the use of artificial tears, which afford only symptomatic relief.

The Use of Dibenamine in the Treatment of Acute Congestive Glaucoma.

William B. Clark and J. Winston Duggan (*American Journal of Ophthalmology*, April, 1951) describe the results of treatment with dibenamine in eleven cases of glaucoma. "Nembutal", one and a half grains, was given thirty to forty-five minutes before administration of the dibenamine, which was given in a dosage of four to five milligrammes per kilogram of body weight dissolved in 300 to 400 millilitres of 5% glucose in physiological salt solution and injected intravenously. The injection was given over a period of one to two hours. This was followed by intravenous administration of 75 to 100 millilitres of 5% glucose in normal saline solution. The patients were confined to bed for thirty-six hours after treatment. The authors state that ten cases were of the acute congestive variety; in seven of these the intraocular pressure of the affected eye was reduced to normal, and in the other three the pressure was reduced, but not to normal. In all the cases in which observation was prolonged, the intraocular pressure began to rise and the symptoms began to return as the blocking action of the dibenamine wore off. In all cases, the other eye, whether it was normal or was the site of chronic primary glaucoma, was little affected by the drug. The authors state that since dibenamine is an effective adrenergic blocking agent, this evidence suggests that excess adrenergic activity is closely related to the cause of certain cases of acute congestive glaucoma. Disadvantages in its use are the need for bed rest in hospital, and intravenous infusion and a relatively high incidence of toxic reactions.

Malignant Glaucoma.

Paul A. Chandler (*American Journal of Ophthalmology*, July, 1951) discusses malignant glaucoma and describes six cases. He states that malignant glaucoma is a form of post-operative glaucoma which takes an unfavourable course and usually results in blindness. The incidence is low and is less in acute congestive glaucoma than in chronic glaucoma. In all cases reported pre-operative examination of the eye showed that the anterior chamber was very shallow and the pre-operative tension high. In most reported cases in which one eye took on a malignant course post-operatively, the outcome was the same in the second eye if it was operated upon. The essential feature of the condition is the forward displacement of the iris-lens diaphragm, completely closing the angle and blocking the wound. The extremely shallow anterior chamber indicates that there is an unusually large lens or a lens disproportionately large in relation to the anterior segment. If the lens is unusually large, it acts as a block between the posterior and anterior chambers. For prophylaxis it would seem advisable to make the tension as low as possible pre-operatively. No case has been reported in which the tension was within the normal range at the time of operation. For treatment posterior sclerotomy has been advocated, but prompt removal of the lens appears to offer the best chance of relieving the condition. If malignant glaucoma ensues in one eye and then glaucoma surgery becomes necessary in the other eye, lens extraction with broad basal iridectomy should be the initial procedure.

Evaluation of the Surgery of Congenital Cataracts.

Frederick C. Cordes (*Archives of Ophthalmology*, August, 1951) reviews the treatment of congenital cataracts. He states that in general surgical measures should be resorted to when the visual acuity is so reduced that the normal use and development of the eyes are impossible. When visual acuity is 20/50 or better, surgical intervention is not indicated. In babies surgical treatment is indicated in those cases in which bilateral complete cataract is present at birth or develops shortly thereafter, and in those in which there is severe bilateral involvement without complete opacification. When there are bilateral dense central opacities and useful vision can be obtained only by dilating the pupil, surgical intervention is also indicated. It is not indicated in those cases in which there is only mild involvement of one eye, even though the other shows extensive opacification. There is difference of opinion as to the age at which operation should be performed. The consensus of opinion is that if cataracts interfere with fixation one eye should be operated on at the age of six months and the second eye should be operated upon in the third year. The visual results from extraction of congenital cataract are much poorer than those following extraction of senile cataract. The ideal operation for removal of congenital cataract would meet the following requirements: it would be a simple procedure, there would be no interference with the pupil, there would be no interference with the posterior capsule and vitreous, and the danger of glaucoma would be minimized. Among operations which are in use, optical iridectomy, although free of complication, does not always give a good result. This operation is not recommended. Discission, in one form or another, is the most popular and widely used operation. The end results are usually good. Discission usually requires a number of operations, and this constitutes a disadvantage from the patient's point of view. Needling

should never be carried out in the presence of a small pupil. If the pupil is small before operation it will be smaller after operation. Complications of discission are cessation of absorption, posterior synechiæ with occlusion of pupil, post-operative irido-cyclitis, organization of capsular tags to form a dense membrane, and glaucoma. Linear extraction is the author's choice of operation. He performs this by making a keratome incision in the cornea, after first injecting adrenaline subconjunctivally. The anterior capsule is ruptured with capsule forceps, lens matter is expressed, and the anterior chamber is washed with saline. If the pupil cannot be dilated, the keratome incision should be at the limbus and a full iridectomy performed. Complications which may occur are detached retina, delayed formation of the anterior chamber, anterior synechiæ and drawn-up pupil, occlusion of the pupil, membrane formation, vitreous loss and glaucoma. If a posterior capsule develops which requires surgical interference, the author believes that the operation of choice is a Wheeler discission. The author stresses the importance of using atropine after any operation for congenital cataract and of continuing its use for two months after the lens has apparently absorbed.

The Ocular Findings of Intracranial Tumour.

M. A. PETROHELOS AND J. W. HENDERSON (*American Journal of Ophthalmology*, October, 1951) review the ocular findings in 358 cases of intracranial tumour. The ocular findings which they analyse are papilœdema, optic atrophy, visual field changes, pupillary abnormalities, and disorders of ocular mobility. Papilœdema was present in 213 cases and was bilateral in 205. Optic atrophy was present in 42 cases, being classified as primary atrophy in 17 cases. Visual field examination was performed in 231 cases, and changes in the fields were noted in 104 cases. Anisocoria was noted in 42 cases. Defects in conjugate movements were noted in 20 cases. Individual muscle palsies occurred in 46 cases, the abducens nerve being involved in 35, the oculomotor in 11 and the trochlear in two, the incidence of nystagmus being 10·9%.

OTO-RHINO-LARYNGOLOGY.

The Effect of Dihydrostreptomycin on the Auditory Mechanism.

ARAM GLORIG (*Annals of Otology, Rhinology and Laryngology*, June, 1951) reports a study of 211 cases of tuberculosis in which treatment was carried out with dihydrostreptomycin to test its therapeutic and toxic effects. Particular attention was paid to the vestibular and auditory mechanisms. The author states that this new streptomycin, on the basis of animal experimentation, was said to be far less toxic than the older preparation. In fact, it has proved to be less toxic to the vestibular mechanism, but far more toxic to the auditory mechanism. In the present series, from one to three grammes of dihydrostreptomycin per day were given for from one to six months. No patient had less than one month's treatment. All patients were tested for vestibular and hearing capacity before treatment was started, and the test was repeated at intervals varying from two to four weeks during treatment and following cessation of treatment. Of the original group of 61 patients 14 developed deafness. The deafness was often severe, being sufficient to be noticeable in conversation. A peculiarity not primarily observed was that deafness sometimes occurred following cessation of the therapy, sometimes after as long an interval as three months. Loss of vestibular function occurred less frequently and after a longer period of treatment, as compared with the changes produced by streptomycin. Studies of hearing loss were made on a further 150 patients, purer forms of dihydrostreptomycin hydrochloride and sulphate being used. The results were similar to those in the earlier group. Forty-seven (31%) showed hearing losses greater than 30 decibels throughout the 500 to 8000 cycles per second range. Loss in hearing rarely became evident in less than three months. In many cases progress continued even after therapy had been stopped. All tests and observations showed the hearing loss to be neurogenic and permanent. In view of these studies it is felt that streptomycin is the better drug, since it is better to lose the vestibular control of balancing than to lose the hearing. The first can be fairly well compensated, but hearing loss is a permanent and serious disability.

Epistaxis.

S. HOROWITZ (*The Journal of Laryngology and Otology*, April, 1951) states that in the ear, nose and throat department of Glasgow Royal Infirmary 100 cases of nose bleeding were studied. There were 59 male patients ranging in age from five to seventy-three years, and 41 female patients, ranging in age from thirteen to eighty-two years. Apart from routine clinical examination to determine the site of hæmorrhage and possible predisposing factors, estimations were made of ascorbic acid levels, prothrombin time and coagulation and bleeding times. In 20 cases both the ascorbic acid level and the prothrombin time were normal. In 70 the ascorbic acid levels were subnormal. In 31 there occurred both an abnormal ascorbic acid level and a prolonged prothrombin time. In 40 a subnormal prothrombin level was found. In 39 subnormal ascorbic acid levels accompanied normal prothrombin times. Studies of age groups revealed that the proportion of patients with both vitamin *C* and vitamin *K* deficiency was highest in the old-age group and lowest in children. The serum calcium content was normal in all cases. The coagulation time was prolonged in seven cases. The bleeding time was normal in all cases. In addition to routine methods of packing and cauterization to control hæmorrhage, patients showing deficiencies were given ascorbic acid 200 to 300 milligrammes daily and 15 milligrammes of vitamin *K* compound for four weeks, with gratifying results.

Tonsillectomy and Adenoidectomy and Poliomyelitis.

O. H. MILLER (*Archives of Otolaryngology*, February, 1951) states that a survey was made of 1229 cases of poliomyelitis occurring in Los Angeles County during the epidemic year of 1949, in order to determine whether there was any relationship between a recent tonsil and adenoid operation and the incidence of poliomyelitis, or the occurrence of bulbar poliomyelitis. Only five of the 1229 patients had had tonsillectomy and adenoidectomy during the thirty days preceding the onset of the disease. It could be calculated from available figures that approximately 15,000 operations of this nature were performed during the year. Statistical calculations place the expected ratio of poliomyelitis at one per 1352 of population. From this figure the expected number of cases amongst the 15,000 patients who had undergone operation would be 11. The actual incidence of poliomyelitis in the members of the city population who had had tonsillectomies and adenoidectomies that year was 10·5. The difference between that figure and the estimated figure of 11 is of no significance, so that it is concluded that the operations had no effect on the incidence of poliomyelitis. Bulbar poliomyelitis developed in only two of the 1229 cases in which poliomyelitis followed operation. It is felt that there is a remote possibility that a recent operation was a factor in development of the bulbar type of disease. There seems to be no reason to discontinue performing indicated tonsillectomies and adenoidectomies during the summer months.

Transtympanic Injection of Anæsthetics for Ménière's Syndrome.

M. S. ERSNER, E. A. SPIEGEL AND M. H. ALEXANDER (*Archives of Otolaryngology*, July, 1951) describe the treatment of Ménière's syndrome by transtympanic injection of anæsthetics. They state that patients for treatment must be carefully selected. A history of labyrinthine vertigo accompanied by tinnitus and subtotal deafness suggests a suitable subject. The middle ear should be healthy with a patent Eustachian tube. After local anæsthetization of the nasal passages a curved applicator bearing a wool plug is passed through the nose to the nasopharynx, and there it is guided under vision through a naso-pharyngoscope, so that the Eustachian tube mouth is securely plugged. The tympanic membrane on the side to be injected is anæsthetized with Bonain's solution or with 10% cocaine in analine oil. With the patient lying on the side one to two millilitres of a solution of cocaine (2%), tetracaine (2%), or alcohol (35% to 50%) are injected through the postero-inferior quadrant of the tympanic membrane. At the end of thirty minutes the applicator is removed from the nose and Eustachian tube. There is no appreciable discomfort. There is usually congestion of the membrane with diffuse redness. After about twenty minutes there develops a sensation of vertigo and deafness. The drop in hearing may last from four to six weeks with gradual improvement. The preoperative vertigo and tinnitus subside when the deafness induced by the injection is complete or profound. Relief from vertigo may be for a long period. No infection or complication resulted from the therapy. Of 37 patients treated, good results were obtained in the majority. Tinnitus decreased in 19 cases, while vertigo completely subsided in 11 and was less in nine cases.

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Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XII.
FENESTRATION AND DIAGNOSIS OF OTOSCLEROSIS IN GENERAL PRACTICE.

IT is extremely easy, even for laymen, to make a fairly accurate guess that a deaf person may be suffering from otosclerosis. Some symptoms rarely occur in other diseases. For instance, if anyone admits having paracusis, a diagnosis of otosclerosis would be almost certainly the correct one. It is sometimes a little difficult to get the patient to understand exactly what one means when one inquires about this symptom, so when I try to elicit it I ask very clearly: "When you are in a loud noise as in a bus, tram or factory, do you hear conversation better than if you were listening to similar speech in a quiet room?"

Otosclerotic patients may not have paracusis, but when they do not, they are usually either in too early a stage to require a fenestration or in too advanced a stage for the operation to help them.

The next point is that the deafness in this disease is very chronic. Further, bone conduction remains remarkably good, considering the degree of air conduction loss. As a result of this the patient, except late in the disease, hears relatively well on a telephone. Tests will show that a 256 vibration per second tuning fork is heard far longer when it is placed on the mastoid than when it is opposite the meatus.

On examination in an uncomplicated case, the drum is within normal limits. However, I have found that otosclerotics are generally far less healthy than the average person, and diagnosis may be complicated by aural sepsis, Eustachian obstruction *et cetera*. Here again the fenestration operation is contraindicated, so not much harm is done if the practitioner fails to suspect the main cause of the deafness. Tinnitus is a reasonably important symptom, for most patients have it at some stage of the disease; but its absence does not exclude otosclerosis.

Other points such as the age of onset are relatively unimportant in a snap diagnosis. Absence of red or ginger hair, absence of quiet speech, absence of gentle mein, absence of a family history of deafness, or of rapid progress of deafness during pregnancy does not at all rule out a diagnosis of otosclerosis, whilst the presence of any such symptoms helps somewhat to substantiate the diagnosis.

The scanty information just given is sufficient for a fairly certain diagnosis of otosclerosis to be made and, once it has been made, the patient should be given the opportunity of seeing a consultant with experience of the fenestration operation for fuller investigation and for advice as to whether operation offers sufficient promise to be worth while in that particular case.

The consultant may advise against operation for various reasons. For instance, if the internal ear proves more than slightly defective, it is little use making a new window through which sounds can reach it. If hearing has fallen below a certain level the operation will not, except in rare "fluke" cases, restore hearing to where an aid may be done without.

Perforations or atrophic scars of the drum or meatal infection are other causes which will exclude operation. Some surgeons will not operate on patients over fifty years old, but most disagree with this and are willing to operate on the dwindling numbers above this age who can stand up to the other required criteria. These are gradually becoming less empirical, but, because of the ones I adhere to at present in my practice, I accept as suitable only about 50% of the patients requesting operation.

Otosclerosis is a common disease resulting in an alteration in the texture of the bone of the capsule surrounding the internal ear. In those cases in which the change progresses to ankylosis of the stapes in the oval window, deafness develops. A similar type of deafness is produced by the fixation of the footpiece of the stapes from any other cause, from closure of the round window, or in those few rare cases reported in which the perilymph turns into a gelatinous substance, or in fact in any condition in which movement of perilymph is restricted.

Normally the impact of sound on the oval window is transmitted through the perilymph up the *scala vestibuli* through the helicotrema at the apex of the cochlea and then down the *scala tympani* to the round window. The movement thus produced in the perilymph in the two scalæ stimulates the auditory receptive mechanism in the cochlear duct from which impulses are sent on to the central nervous system.

In the conditions referred to, where the perilymph is prevented from moving, a syndrome is produced which I call "perilymphatic stasis", that is, paracusis with deafness for air conduction, coupled with relatively little or no loss of hearing for bone conduction. In the later stages of otosclerosis, degeneration of the cochlea develops for reasons which are still somewhat debatable.

In otosclerosis with a healthy cochlea, except in the rare cases in which the round window is also obstructed, hearing can be restored by making an opening into any part of the perilymphatic space of the vestibule or its extensions. The most convenient place for doing this is the external semicircular canal, as it is here possible to cover the artificial opening with a thin flap graft which will protect it from infection.

The main technique of the operation is directed towards making the opening without traumatizing the delicate membranous semicircular canal (either by instruments or by the heat from the burrs used) and towards removing traces of bone chips and bone dust and placing the opening in that part of the canal where subsequent bony closure is least likely to occur. The flap graft consists of that part of the meatal skin immediately above Shrapnell's membrane, which was estimated in a recent article as being only one-tenth of a millimetre thick.

The operation usually produces temporary vertigo, but this is reduced by certain techniques. The most annoying feature of the operation is that dressings have to be continued frequently for a somewhat lengthy period afterwards, though with good methods in few cases are dressings required for as long as three months, and with skin graft techniques some wounds are dry at the end of twenty-eight days.

The auditory improvement is phenomenal in the best cases, hearing not infrequently improving from four inches to twenty feet for the conversational test voice.

Unfortunately the initial improvement is not always maintained, and the new fenestrum is said to close in 20% of cases, usually between the fourth and ninth months after operation, though I think this percentage has been overestimated. Results vary, naturally, in different operators' hands.

Most claim a permanence of the fenestrum with satisfactory hearing in over 60% of cases, though some claim up to 80%, a level probably compatible only with exceptional skill or unbridled optimism. A few patients, probably 2% to 5%, hear worse, either from accidental trauma, from infection, or because of an acceleration of coexistent mild cochlear degeneration, which may be present in the less favourable cases acceptable for operation.

Patients, whose hearing is not improved or is actually made worse, prove surprisingly tolerant of their misfortune, probably because the fenestrator nearly always selects for operation the deafer ear, which is not being used anyway.

The main advantage of having the operation over the wearing of a hearing aid is that the use of an aid does nothing to stop the progress of the deafness and requires constant expense for upkeep; also with certain patients such aids produce a sense of inferiority in spite of their obvious usefulness.

Summary.

1. Stress is laid on how otosclerotics can be suspected with tolerable certainty.

2. The principle of the fenestration operation is simply explained.

3. A rough idea is given of what may be expected from fenestration.

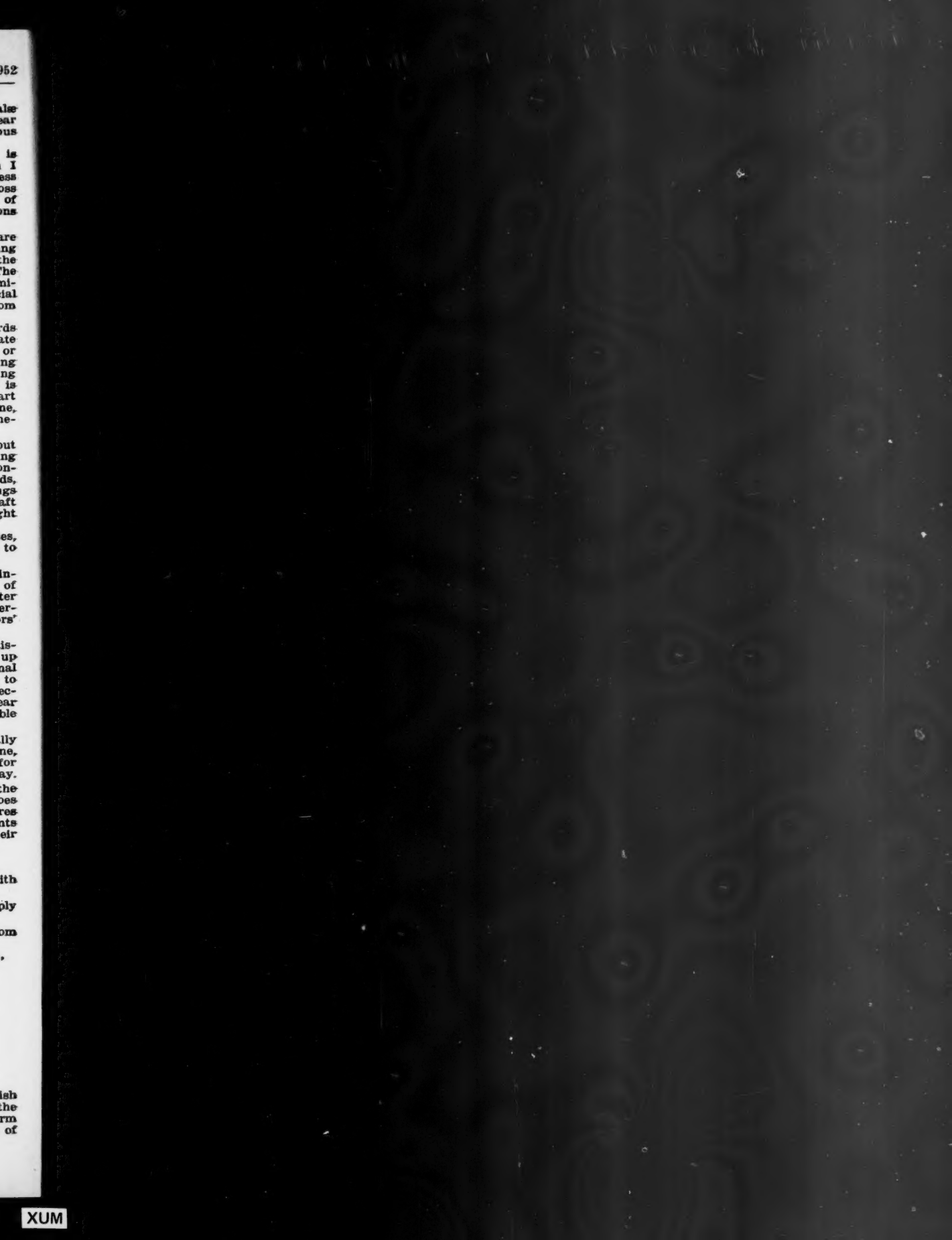
A. B. K. WATKINS, M.S. (London), F.R.C.S. (England).

Newcastle,
New South Wales.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 6, 1951, at the Saint George Hospital, Kogarah. The meeting took the form of a number of clinical demonstrations by the members of



the honorary medical and surgical staffs of the hospital. Parts of this report appeared in the issues of March 8 and 15, 1952.

Thrombosis of Inferior Mesenteric Vein.

DR. B. DENNING presented a male patient who had been first admitted to hospital on November 27, 1950, with the presenting symptoms of swelling and tenderness of the left leg for five days. The patient had been treated for congestive cardiac failure for the previous three months. Swelling and pain of the left leg had been present for five days with restricted movement. Examination of the patient revealed pronounced œdema of the left leg up to the knee. The leg was very tender to touch, especially behind the popliteal fossa. A provisional diagnosis was made of thrombosis of the popliteal vein. Heparin therapy was given for forty-eight hours and then suspended. Dicoumarol therapy was recommenced on December 6. The prothrombin time was reduced to 64%. The thrombus in the leg almost completely disappeared, although slight induration was still present in January, 1951. The patient was discharged from hospital on January 5.

On his second admission to hospital on February 15, 1951, he complained of tightness in the abdomen for half an hour after eating some figs. The pains soon became worse, and when the patient went to defæcate he passed blood *per rectum*. The bowel actions soon became irregular and more frequent, and much blood was passed *per rectum*. Pains, which had began across the lower part of the abdomen, soon shifted to the upper part of the abdomen. Examination of the patient revealed deep tenderness with superficial leg paræsthesia across the mid-abdomen just below the umbilicus. Rigidity and guarding were evident. After rectal examination a slight amount of blood was present on the examining finger. The abdomen had a very distended, bloated appearance. Operation revealed thrombosis of the inferior mesenteric veins involving the supply to the whole sigmoid colon and extending down to the rectum. Colostomy was performed. After operation the patient was given a light fluid diet by mouth and had six-hourly gastric aspirations. The colostomy opened the day after operation. Phthalylsulphathiazole lavage was performed two-hourly into the distal segment of bowel. The patient's general condition was then fair. Two days later anticoagulant therapy was commenced. On the following day he passed some mucus with much flatus *per rectum*; it was heavily blood-stained. The abdomen was swelling up, but the condition was relieved by clearing out the distal drainage tube. The patient was given further anticoagulant therapy for forty-eight hours. When bleeding occurred about the colostomy wound, heparin therapy was suspended. Ten days after operation the patient was allowed to get up. Two weeks later he was well and was discharged from hospital, being told to return later for barium enema examination.

On his third admission to hospital, on March 16, 1951, he complained of gripping pain in the abdomen for six hours. The pain was colicky in nature and no flatus or fæces had been passed from the colostomy since its onset. The patient was very distressed and was rolling about the bed with pain. His tongue was clean and moist. There was no rigidity of the abdomen, but generalized tenderness was present with turbulent borborygmi. The colostomy was dry and clean; no abnormality was palpable. Morphine was ordered, with bowel lavage. On the next day distension was less and the pain had ceased. The patient was vomiting dark coffee-ground material. The colostomy was not acting, so pressure lavage was given, and the colostomy worked one hour later. Two days later it was functioning well. A blood count revealed a total of 6500 leucocytes per cubic millimetre, made up of neutrophile cells 76%, eosinophile cells 2% and lymphocytes 8%. The fasting blood urea content was 56 milligrammes *per centum*. Colostomy lavage was given under pressure with a fæcal return. On March 27 intravenous saline drip therapy was commenced. There was then no rigidity or distension of the abdomen. Heparin therapy was commenced and continued at intervals according to the blood coagulation time. Intravenous fluid therapy was suspended on March 30. On April 14 a barium enema examination was performed. The barium ran from the rectum through the colostomy. Early diverticulosis was detected in the left side of the colon. On April 26 the patient was discharged from hospital. The final diagnosis was infarction of the large intestine due to thrombosis of the mesenteric artery.

Carcinoma of the Breast.

Dr. Denning's second patient, a woman, aged fifty-one years, had been admitted to hospital on March 21, 1949, with the history of lump in the breast for nine months. It was not tender, and there was no discharge from the nipple. A course of deep X-ray therapy three months previously had had good effect, the swelling being greatly reduced in size. The tumour, which was above and medial to the nipple of the left breast, was hard, painless and mobile. Left radical mastectomy was performed on March 22. During convalescence the wound became infected and an abscess in the breast was incised. The patient was discharged from hospital on April 26. On January 9, 1950, a small lump was detected in the line of the operation scar. X-ray treatment was given to the chest on April 17. No change occurred in the lump. Further X-ray treatment of the chest was carried out in June, 1950. One month later the lump had decreased in size and almost disappeared. In October, 1950, the patient felt well. She continued attending the out-patient department at two-monthly intervals, and at the time of the meeting was feeling well. The lump had quite disappeared. The final diagnosis was carcinoma of the breast.

Bilateral Cup Arthroplasty.

DR. A. L. WEBB presented a woman, aged sixty years, who had been admitted to hospital on January 21, 1951, complaining of pain in both hips for approximately ten years. The pain was followed by stiffness in both hip joints and then by limitation of movement in all directions. For six years the patient had had to walk with crutches. On examination of the patient the hip joints were found to be held in flexion adduction and internal rotation. Movement was very limited. At operation the patient was found to have flattening of the femoral head, dense adhesions about the left hip joint and roughening and widening of the rim of the acetabulum. Cup arthroplasty was performed on the left hip. The patient was given one litre of blood and recovered well from the operation. A Steinmann's pin was inserted through the tibial tuberosity and extension was applied, a Braund's frame being used. The patient's progress was satisfactory, and she was discharged from hospital on March 16, walking on crutches. She carried on with physiotherapy as an out-patient. She was readmitted to hospital on August 19. The previous operation wound was clean. Operation was performed on the right hip on August 21. The condition found was osteoarthritis of the right hip. A cup arthroplasty was performed. After similar post-operative care, the patient's progress was satisfactory and she was discharged from hospital on October 14.

Osteochondritis Dissecans.

DR. C. P. HUDSON showed a male patient, aged twenty-nine years, suffering from *osteochondritis dissecans*. He had been a patient in the hospital in June and July, 1951, with multiple fractures of the left femur, the left radius and the left ulna, which were plated. As an out-patient he was given physiotherapy and the injuries responded well. He complained of persistent pain in the left knee, and X-ray examination revealed *osteochondritis dissecans*.

Osteochondritis of Epicondyles.

Dr. Hudson showed a male patient, aged thirty-one years, suffering from osteochondritis of epicondyles. He attended as an out-patient on November 8, 1951, complaining of tenderness of the right elbow of three months' duration. No definite injury had been sustained prior to the symptoms. Examination of the patient revealed tenderness of the lateral epicondyle and pain on passive stretching or active contraction of the extensor muscles. X-ray examination revealed a small area of *osteochondritis dissecans*. By November 29 the symptoms had become worse, and the patient was unable to use his right arm owing to pain. He was to be admitted to hospital for operative treatment.

Fractured Tibia and Fibula.

Dr. Hudson's third patient was a male, aged forty-three years, who had been admitted to hospital on April 4, 1951, with a nine days old compound fracture at the right ankle, sustained when he fell under a motor-car. He had been treated at another hospital with penicillin and tetanus antiserum. The limb was manipulated and put into a plaster splint at that hospital, but the fragments slipped out of position.

On his admission to hospital his leg was in plaster and causing him continual pain. There was no blueness, coldness, numbness or severe pain from the toes. At operation on April 9 a Kirschner wire was inserted into the calcaneus, and the fracture was reduced under general anæsthesia. The leg was put up in traction in a Braud's splint. The patient was discharged from hospital on May 25 with his leg still in plaster, and walking on crutches. He was examined in

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the out-patient department, where an X-ray examination revealed that the position of the fracture had deteriorated and that there was no evidence of union. He was readmitted to hospital on July 29. At operation on July 30 the condition found was an ununited fracture of the tibia, and a sliding graft was put down the tibia. The leg was placed in a full leg plaster cast. It was left in plaster until September 12, when it was removed and the sutures were taken out. He was discharged from hospital on September 13 on a walking caliper. Since then the union and his progress had been satisfactory.

Dr. Hudson's fourth patient had been injured in August, 1949, and had been admitted to hospital with a fracture of the left tibia and fibula, and concussion. He responded only slightly to painful stimuli. On examination of the patient, his pupils were fixed and unequal at first, but they soon became equal and reactive. Extensive laceration and contusion of the lower third of the left leg were present with protrusion of fragments of the fibula through the lateral aspects of the leg and of fragments of the tibia through the medial aspect of the leg. The fractures of the tibia and fibula were plated in the operating theatre on August 20, and the leg was put in a plaster cast. He was discharged from hospital on April 16, 1950, with poor granulation over the site of plating. He was readmitted to hospital in December, 1950, and the plate and pin were removed. The bone edges were freshened by drilling, and the plaster cast was reapplied. He was readmitted to hospital for multiple skin grafts and a bone graft to the tibia. Since then he had been treated as an out-patient in a plaster splint and had been walking on crutches.

Severed Brachial Artery.

Dr. Hudson next showed a male patient, aged seventeen years, who had been admitted to hospital on August 25, 1951, having fallen on his outstretched left arm whilst playing football.

On examination of the patient, his left hand was blue and he was unable to move his fingers. The swelling extended half-way up the forearm. There was also a large hæmatoma around the elbow joint. Sensation was lost over the lower third of the arm and hand. X-ray examination revealed a posterior dislocation of the olecranon on the humerus. The dislocation was reduced under general anæsthesia in the casualty department. He was then taken to the operating theatre, where the elbow was explored and the brachial artery was found to be severed, and tied. On August 27 the hand was warm and the radial arterial pulsation was just palpable. Sensation was lost over the ulnar nerve distribution, and the patient was unable to extend his fingers. On August 28 sensation had improved, but the radial pulse was not palpable. The hand was warm. On August 30 his fingers were cold, but sensation was still improving. On August 31 the radial pulse was again palpable, but sensation was unaltered. He still had no ability to extend his fingers. On September 1 movement was improved, but sensation was unimproved. On September 3 sensation was improving in the ulnar distribution on the hand. Movement had improved, but still there was no extension at the metacarpo-phalangeal joints. On September 4 a cock-up splint was applied, and physiotherapy to increase the movements of the elbow joint were commenced. The position remained unchanged till his discharge from hospital on September 18, after which he continued physiotherapy as an out-patient.

Prolapsed Intervertebral Disk.

Dr. Hudson finally showed a male patient, aged fifty-four years, who on November 22, 1951, complained of low back pain of the right sciatic nerve distribution of seven weeks' duration. He had sustained no trauma apart from that due to gardening; the pain was constant and growing worse, and was interfering with his sleep. He had had to have morphine to get relief. On examination of the patient there was evidence of pressure from a prolapsed lumbar sacral disk on the right first sacral nerve root, with wasting of the back of the thigh and paræsthesia in the first sacral dermatome. The ankle jerk was absent. Consistent X-ray findings were obtained. At operation, the first sacral nerve was tightly stretched over a herniated prolapsed intervertebral disk (between the fifth lumbar and the first sacral vertebra). The disk was removed by an interlaminal approach.

Tubal Occlusion Cured by Myomectomy.

DR. K. S. RICHARDSON showed X-ray films to indicate the results achieved by conservative myomectomy. The patient, a married woman, aged thirty-five years, with one child, was examined because of secondary sterility. Hysterosalpingography revealed complete Fallopian tubal occlusion, with apparently a symmetrical fibroid tumour of submucous type. This was confirmed at operation. The second hysterosalpingogram had been taken six weeks after operation. It showed the tubes well outlined, and an excellent seven-hour intraperitoneal "spill".

Pregnancy following Salpingostomy.

Dr. Richardson then showed a married female patient, aged twenty-five years, who had been first examined in July, 1950, when she complained of primary sterility since her marriage two and a half years previously, and of dyspareunia for the same period. She gave a history of having had some acute abdominal pain at the age of seventeen years, and was treated in a country hospital under observation for ten days. Vaginal examination revealed a small anteverted uterus with a large tender mass in the posterior fornix, pressure on which produced the pain of intercourse.

Section was performed in August, 1950, and the mass was found to be composed of both Fallopian tubes and ovaries, which were all securely bound down in the pouch of Douglas. The structures were freed and on each tube an everting cuff salpingostomy was performed. Convalescence was uneventful. A search was made for the appendix, but none was found. It would appear that the previous abdominal crisis caused a sloughing of this organ with formation of the pelvic inflammatory mass. A hystero-salpingogram prepared after operation revealed good intraperitoneal "spill". The patient at the time of the meeting was thirty-two weeks pregnant, and the progress was normal.

Bilateral Endosalpingioma Associated with Pregnancy.

Dr. Richardson finally showed a married woman, with three children, who had been first examined in June, 1951. Her last menstrual period had occurred on April 11, 1951, and she considered that she was pregnant. Abdominal examination revealed a large mass below and to the left of the umbilicus, and the uterus was thought to be about the size of a ten weeks' pregnancy. The vaginal findings confirmed this, and there seemed to be also a mass in the right fornix. The Aschheim-Zondek test produced a positive result. After her admission to hospital she had some heavy bleeding, which settled down after about six days. Section was then performed and a tumour was found and removed (the tumour was shown at the meeting). In view of possible malignancy, panhysterectomy was performed. The patient's convalescence was uneventful.

A pathological examination of the uterus and both Fallopian tubes and ovaries was carried out. The uterus measured 9·0 by 6·0 by 5·0 centimetres. It had many dense adhesions on the posterior surface. Both ovaries had been replaced by tumour masses. The right (larger) was made up of a cyst 10 centimetres in diameter, which appeared to have solid portions to it, and from which protruded a partially solid and partially papilliferous growth 7·5 centimetres long by 3·5 centimetres broad and 3·5 centimetres thick. The left ovary had been replaced by three cysts of approximately equal size, 4·5 centimetres in diameter. It also appeared to have solid components. Examination of the uterine wall revealed that the patient had probably had a recent abortion, which might have been complete. There was a well-developed decidual reaction in the endometrium, there were a number of large blood spaces and masses of fibrin, but no actual villi were seen. The cervix appeared normal. Examination of sections of the right ovary revealed that the tumour was an endosalpingioma; it occurred as a series of cysts lined by papillary ingrowths, which were covered by columnar "tubal" type epithelium. The connective tissue might be fibrous, but in many areas it had undergone myxomatous change and appeared to produce pinkish substance, which had filled many of the cysts with a mucinous or jelly-like material. Calcified "psammoma bodies" were seen in the walls of the cysts. The tumour of the left ovary was identical in nature. Although there was considerable epithelial hyperplasia, no malignant change was seen.

Tabes Dorsalis.

DR. S. F. MARSHALL showed a male patient, who had reported to hospital on August 14, 1951, with shooting pains in the legs. He gave a history of genital sore and asymptomatic bubo in 1917, and the Wassermann and Kahn tests had produced positive reactions on July 25, 1951.

On examination of the patient, his left pupil was larger than the right, and reacted slightly to light and accommodation. The knee jerks and ankle jerks were absent, and Rombergism was pronounced. On August 28 the cerebro-

spinal fluid reacted to the colloidal gold test with a typical paretic curve ("5555542100-0"); Pandy's test revealed an increase in the globulin fraction; the protein content was 100 milligrammes per 100 millilitres; mononuclear leucocytes numbered one per cubic millimetre and erythrocytes six per cubic millimetre; the fluid gave a positive reaction to the Wassermann test and to the Kahn test.

The patient was treated with bismuth, two millilitres twice in one week, then once per week, and with procaine penicillin, 1,200,000 units three times per week for four weeks to a total of 14,400,000 units.

On October 26 the treatment had been completed, and the Rombergism had decreased.

On November 30 the pains were practically absent.

Lichen Sclerosus et Atrophicans.

DR. G. CHAMBERS showed a female patient, aged eleven years, who for five years had been developing white "spots" on the chest, back, neck and arms, and a few on the thighs. The lesions were about 0·2 to 1·0 centimetre in diameter and pure white in colour. There was some stippling in the centre of each lesion. The lesions were atrophic, and the condition was regarded as *lichen sclerosus et atrophicans*. Some improvement had taken place since a daily dose of 30,000 units of vitamin *A* had been instituted.

Nævus Unius Lateris.

Dr. Chambers then showed a female patient, aged eighteen years, who for the past six years had had a light brownish lesion on the left side of her neck just below the mandible. It had remained unchanged until recently, when there appeared to be some increase in extent. The lesion took the form of discrete circinate areas about 0·2 to 1·0 centimetre in diameter, the centre being paler than the margin. On the mid-line over the thyroid cartilage there was a double tone of similar colour about 0·2 centimetre wide and about 4·0 centimetre long, separated by about 0·2 centimetre of normal skin. The condition was regarded as *nævus unius lateris*.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

ASSISTANT SURGEON REDFERN TO GOVERNOR MACQUARIE.[1]

Sydney, October 18, 1819.

Sir,

His Royal Highness the Prince Regent having been pleased to appoint Mr James BOWMAN to be Principal Surgeon in His Majesty's Territory in New South Wales in room of D'ARCY WENTWORTH Esquire resigned. And as I had reasonable expectations, from the strong recommendation of Your Excellency in my favour, from a long, laborious, and useful service of eighteen years: and from the promise of Earl BATHURST to Viscount CASTLEREAGH and from his Lordship to my brother that I should succeed to the vacancy occasioned by Mr Wentworth's resignation, I cannot but feel that these reasonable expectations have thus terminated in severe mortifying disappointment—that my most sanguine hopes and best prospects in life are thus utterly blasted. I have, therefore, the honour of thus tendering my resignation—soliciting your Excellency to have the goodness to forward the same for the gracious acceptance by His Royal Highness, the Prince Regent, and at the same time beg leave most respectfully to entreat that your Excellency will be pleased to permit me, in the meantime, to retire from the duties of my situation as Assistant Surgeon, till the will and pleasure of His Royal Highness on the subject shall be made known. Your Excellency's most obedient servant,

W. REDFERN,

Assistant Surgeon to His Majesty's Settlement in New South Wales.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

SIR RICHARD STAWELL'S LETTERS.

SIR: The "Sir Richard Stawell Oration" trustees are desirous of collecting as many personal letters of the late Sir Richard Stawell as possible to enable future orators an opportunity to study them. He possessed so many fine personal qualities which would be unknown to orators except by tradition. A collection of these (or copies of them) would be invaluable. An appeal is therefore made to the numerous students, resident medical officers and practitioners throughout Australia who were associated with Sir Richard and who may have kept and treasured letters from him.

Will the practitioners who have them kindly forward them to the Chairman of Trustees, who will gratefully acknowledge them?

Yours, etc.,

A. E. ROWDEN WHITE,
Chairman of Trustees of the "Sir Richard Stawell Oration".

14 Parliament Place,
Melbourne,
February 29, 1952.

TUBERCULOSIS AND THE GENERAL PRACTITIONER.

SIR: The excellent paper by Dr. P. S. Woodruff on "Tuberculosis and the General Practitioner" (THE MEDICAL JOURNAL OF AUSTRALIA, February 9, 1952) under the heading of "Rehabilitation" mentions Bedford Industries. It omits what I believe has up to the present been a more significant though less publicized rehabilitation scheme, namely, the rehabilitation of patients at Kalyra Sanatorium by the transfer of suitable patients to the staff. Such patients have numbered up to three-quarters of the entire staff. Not only has this scheme rehabilitated a greater number of full-time workers as distinct from those who merely supplement their pension by the allowable £2 per week, but the work itself is of vastly greater significance in combating tuberculosis, as it is directly concerned with the treatment of the tuberculous—unquestionably a more worthy object than the manufacture of washboards, however desirable these may be.

During the war and early post-war period other sanatoria in South Australia had many empty beds because of the difficulty of obtaining staff. Kalyra Sanatorium, staffed largely by former patients, was in a much more favourable position and was able to help the tuberculosis services out of an extremely difficult position.

Working under close medical supervision, the ex-patients have done well, particularly as it has been possible to lighten their work a little when necessary. They have been picked people, and have always been among our most valuable employees. No public subscription has had to be raised for their employment, and the value of their work is to some extent reflected in the fact that daily average costs per patient at Kalyra have consistently been lower than those of local sanatoria employing only non-tuberculous labour.

This field of rehabilitation is limited, but it is an exceedingly valuable one, and has to my mind not been used widely enough. It is not spectacular, but to the discerning it is extremely interesting. Without publicity or subsidy it has done and is doing work of very considerable value.

Yours, etc.,

O. E. NICHTERLEIN, M.A., M.D.,
Medical Superintendent, Kalyra Sanatorium.

Belair,
South Australia,
February 25, 1952.

ORTHOPÆDIC ASPECTS OF RHEUMATOID ARTHRITIS.

SIR: In my paper "Orthopædic Aspects of Rheumatoid Arthritis" read before the New South Wales Branch of the British Medical Association on August 30, 1951, and published in THE MEDICAL JOURNAL OF AUSTRALIA on November 3, 1951, I neglected to make due acknowledgements for the photographs used to illustrate the text. Will you kindly allow me to rectify this error by publishing this letter in your next issue?

For Figures VIII and X I am indebted to the "Textbook of the Rheumatic Diseases", by W. S. C. Copeman (E. and S. Livingstone, Limited, Edinburgh, 1948), and for the

remainder to the "Manual of Rheumatic Diseases", by W. P. Holbrook (D. F. Hill, The Year Book Publishers, Inc., Chicago, 1950).

Yours, etc.,

135 Macquarie Street, Sydney,
February 19, 1952.

GORDON COLVIN.

SURGERY OF THE ADRENAL GLANDS.

SIR: Mr. Edward Wilson has presented a concise and instructive account of surgery of the adrenal glands. However, his apparent advocacy of partial adrenalectomy for the patient with hirsutism and mild virilism must be challenged, lest it lead to more widespread use of this mode of treatment for these unfortunate women. Hirsutism in the post-pubertal female may be considered under three headings, if one excludes the excessively rare virilizing ovarian tumours.

1. Hirsuitism with overt masculinization due to an adrenal neoplasm. Amenorrhœa is invariable and the excretion of 17-ketosteroids is very high. Surgical removal of the tumour gives a gratifying cure.

2. Hirsutism with overt masculinization due to adrenal cortical hyperplasia. Amenorrhœa, again, is almost invariable, as are the changes in configuration of the body, in the development of the muscles, and in the voice. Excretion of 17-ketosteroids is moderately to greatly increased. In these patients partial adrenalectomy yields poor results. The analogy to thyroidectomy for hyperthyroidism is fairly close. If too little gland is removed the remainder rapidly regenerates; if too much is removed permanent insufficiency may result. It is possible to abolish adrenal hypersecretion by radical subtotal adrenalectomy as described in a recent report (Priestley *et alii*, 1951), but this involves total unilateral adrenalectomy and removal of 90% of the remaining adrenal gland. Such a calculated risk is probably justified in the treatment of Cushing's syndrome, but rarely so in the treatment of virilism.

3. Hirsutism with no, or minimal, masculinization. Such patients are seen far more frequently than are those in the first two categories. Most of them have familial hairiness or hyperactivity of hair follicles due to abnormal "end-organ sensitivity" to a normal amount of circulating androgen. Those patients with levels of 17-ketosteroid excretion a little above normal may, perhaps, have slight adrenal hyperactivity, but, more probably, have some disturbance of steroid metabolism that is as yet undiscovered. In most endocrine clinics it is thought unjustifiable to operate on this type of patient in order to achieve a dubious cosmetic result.

Yours, etc.,

149 Macquarie Street, Sydney,
February 23, 1952.

K. S. HARRISON.

Reference.

Priestley, J. T., Sprague, R. G., Walters, W., and Salassa, R. M. (1951), "Subtotal Adrenalectomy for Cushing's Syndrome", *Annals of Surgery*, Volume CXXXIV, page 464.

SIR: I read with interest the article by Wilson on "Surgery of the Adrenal Glands" (February 23, 1952) and feel that some comment may be made about the indications for adrenalectomy, either one gland being removed or parts of each gland. With the operative indications under the heading of adrenal tumours, it is obvious that these conditions must be tackled, if diagnosed, by radical surgical measures in view of our existing limited state of knowledge.

However, as far as Cushing's syndrome is concerned, where the ætiology and mechanisms through which the disease is mediated are completely unknown, I do not feel that surgical procedures are justifiable. As regards virilism and hirsuties, it is pertinent to point out that the adrenal glands have quite an extraordinary regenerative capacity and, on experimental grounds alone, one could predict with reasonable accuracy that removal of one or large amounts of both adrenals would be followed by regeneration and the establishment of the clinical situation which existed prior to operation. If an adrenal cortex is so-called hyperplastic, it will finally and rapidly get back into the same state after operation as it was before. The normal pituitary stimulus, whether it be growth, somatotropic or some other undiscovered or unpostulated factor, is still operating. The ultimate answer, if one would venture to look ahead, probably lies in pituitary or adrenal cortical blocking or competitive agents.

From a practical point of view, there still remains the immediate question of dealing with the problems these patients present, and surely it would be preferable to get psychological readjustment and the acceptance of simple measures (compare Molesworth (1951) on hypertrichosis) rather than submission to a hazardous operation of unpredictable results.

Yours, etc.,

The University of Sydney,
Sydney,
February 27, 1952.

GEORGE READ.

Reference.

Molesworth, E. H. (1951), "An Introduction to Dermatology", J. and A. Churchill.

THE CONSERVATIVE TREATMENT OF PERFORATED PEPTIC ULCER.

SIR: Your leading article of January 12, 1952, on the conservative treatment of perforated peptic ulcer seemed to indicate that the method has not yet been used to any extent in Australia.

It might be worth recording my limited experience in this regard, in the course of general practice over the past four years. During this time I have treated eight cases of ruptured gastric ulcer in men only. Five of these patients were treated by immediate surgery (oversewing), and two of them (one, twice) by conservative means (intravenous fluids, morphine, antibiotics and Meulengracht diet).

In the three cases in which conservative treatment was given, recovery ensued and subsequent barium meal X ray revealed positive evidence of ulcer. I was encouraged to avoid surgery on these three occasions by the following considerations: (*a*) Good results published from England in reducing the mortality rate by this means (as high as 25% following operation, for example, an extensive series from Adelaide, published since the war); this gave me moral support instead of a sense of neglect in failing to operate. (*b*) On the three occasions I was reasonably sure that the stomach was fairly empty of solids at the time of rupture, and equally certain of the presence of alcohol there. (*c*) An adverse family history of one of the patients, whose two brothers had died following or shortly after operations for ruptured gastric ulcer. At least the result could be no worse here. (*d*) Conservative treatment had succeeded once before in the other patient and was expected to do so once again. Subsequent barium meal in this case showed a good number of adhesions present—rupture had followed the drinking of two glasses of beer after a spell of practice rowing.

All of the five cases subjected to operation recovered, though two of them, in the older age group, two years later developed a further ulcer and required medical treatment.

It seems that either method of treatment, surgical or conservative, is successful, if proper facilities are available, and one's clinical sense in choosing which method to use is either good or lucky.

Yours, etc.,

Penrith, New South Wales,
Undated.

W. F. J. CAMMACK.

WORLD HEALTH ORGANIZATION.

SIR: In its programme of promoting improved medical education throughout the world, the World Health Organization has been asked by several countries (among them Pakistan, India, the Philippines, Iran and Singapore) to obtain professors for two or more years in anatomy, physiology, pharmacology and pathology. There is also a position open as principal of a medical college in India, for which persons with experience in the administration of medical schools are eligible. Persons interested in any of these positions may write directly to the World Health Organization, Geneva, Switzerland, for further details.

Yours, etc.,

JAMES L. TROUPIN, M.D.,
Chief, Section of Assistance to Educational Institutions.

World Health Organization,
Geneva,
Switzerland.
February 5, 1952.

Obituary.

BRUCE MAITLAND CARRUTHERS.

WE are indebted to Dr. T. R. Gaha for the following account of the late Dr. Bruce Maitland Carruthers.

Bruce Maitland Carruthers, O.B.E., was born at Maitland, New South Wales, on April 23, 1892. He was educated at Fort Street High School and Newington College, and qualified at the University of Sydney. He enlisted in the Royal Army Medical Corps in April, 1915, soon after graduating, and saw service in France, England, India, Salonika and the Dardanelles. After some post-graduate work in England, he returned to Australia, and became assistant director at "Russell Lea" Red Cross Home, Sydney, for the treatment of psychiatric patients.

My first meeting with him was in 1928 when he was my senior house surgeon at the Royal Hobart Hospital. He was a very fine medical officer and a friend. He taught me not only what I know of the medical art, but also its human approach. His quality can perhaps be illustrated by an example. A nurse—the daughter of a poor family—died. He undertook all the funeral arrangements including the financial obligations. In the second World War he enlisted as a captain in the Australian Army Medical Corps on July 1, 1940. Soon after arrival in Palestine, he was appointed Assistant Director of Hygiene of the First Australian Corps. During 1941 he saw service in Tobruk and Greece. On December 30, 1941, he was awarded the O.B.E. for his services in a military and civil capacity. He was returned to Australia in April, 1942, promoted to the rank of lieutenant-colonel and appointed Assistant Director of Hygiene of the First Australian Army. Later he was appointed Assistant Director of Medical Services in Tasmania Command. He was discharged from the army on August 28, 1945.

His association with the Public Health Department in various phases lasted about sixteen years. He was appointed administrator of Lachlan Park Mental Hospital in 1935. In 1936 he was appointed Director of Public Health for the State of Tasmania, and this covered Lachlan Park administration. He still held this position when he enlisted in 1940. Whilst holding this job he was largely instrumental in successfully negotiating the arrangement of "free" medical services to country districts. After discharge from the army in December, 1945, he was appointed Director of Hospital and Medical Services for the State of Tasmania. In May, 1951, he was appointed Director of Public Health and Director of Hospital and Medical Services, which post he held until his death. Only to those who know the full ramifications of the various appointments will come a full realization of the work involved and long hours of overtime, frequently extending into week-ends. The Royal Hobart Hospital was his particular interest, and a very large share of the credit for its present standing in the community and its service to the public as an entirely free hospital must go to him. He was ever ready to help in any good cause, but particularly so to a person whose finances did not permit a claim on perhaps the best that was available medically.

That his contributions to medicine in Tasmania were widely appreciated was demonstrated in the innumerable inquiries during his last illness. His passing was mourned by all. Those who knew him best appreciated his great contribution to the public service he took such pleasure in successfully performing.

Dr. Frank W. Fay writes: Bruce Carruthers was a man much respected and admired by his colleagues, particularly for his administrative ability, his honesty of purpose and that rare capacity of, once having started on a project, seeing it through to a successful conclusion. During the negotiations in 1928 and subsequently for the reintroduction of the honorary staff to the Royal Hobart Hospital, he and I had innumerable meetings and discussions. His judgements were always sound, his suggestions were fair and moderate, and his advice was wise and temperate. He played an important part in bringing these negotiations to a successful conclusion. Again, as Assistant Director of Medical Services for Tasmania during World War II, he automatically became chairman of the Tasmanian Medical Coordination Committee. In this capacity his broad outlook, happy smile and knack of turning major problems into minor very soon commanded the respect and affection of the whole committee. Bruce loved a party (so do I, and so do you), but this never interfered with his work, and he was always one of the first on duty at the Public Health Department. Of criticism, one would say that as Director of Public Health he tried to do too much. He was prepared to shoulder all responsibilities, not only of the director himself, but also of all officers of the department down to the most junior clerk. He has, however, left behind him an extremely well-organized department of public health, and one which has made history by its introduction of free medicine for outlying country districts. Bruce Carruthers, as chairman of the board of the Royal Hobart Hospital, did a grand job in establishing the honorary medical advisory committee. This committee has worked in amicable liaison with the board for many years. Bruce has left behind many friends and admirers. His untimely passing was a sad blow both to his friends and to the various institutions and departments for which he was responsible. Tasmania can ill afford to lose a man like Bruce Carruthers.

Dr. W. W. S. Johnston writes: In the Middle East as officer commanding 2/3 (Corps) Field Hygiene Section, Major B. M. Carruthers rapidly established a high reputation by his efficiency and energy. During the Libyan campaign he and his unit carried out very important work in the organization of hygiene, a matter of considerable difficulty, in view of the widespread territory and the rapidity of movement involved. He was subsequently appointed Deputy Assistant Director of Hygiene of First Australian Army Corps, commencing duty immediately prior to the embarkation for Greece. Here his ability and technical knowledge were of the greatest value in putting into effect the arrangements designed by Major-General Burston and Brigadier N. H. Fairley at Australian Imperial Force Headquarters to combat disease in that area. This campaign was too short-lived to demonstrate fully the excellence of his planning, but he had the opportunity of displaying this more adequately in the Syrian campaign, which followed a few weeks later. The fighting was bitter, the terrain difficult, and although the dangers from malaria were realized, the area was uncharted as regards the prevalence of the disease. In addition the troops were untrained in the antimalarial measures laid down and the officers largely sceptical of the benefits of the discipline prescribed. It was still a long way to the days in the South-West Pacific when an individual conscience had developed and neglect of precautions was regarded as a crime. But Carruthers successfully overcame such obstacles. He was indefatigable both in implementing the general plan and in personally supervising details. Much of the success against disease in Syria was due to his personal energy and example. He had the gift of logical

persuasion when discussing medical problems with senior combatant officers and an infectious enthusiasm that stimulated those with whom he worked. After his return from the Middle East he was appointed Assistant Director of Hygiene of First Australian Army with the rank of lieutenant-colonel. Later in 1942 he was transferred to Tasmania as Assistant Director of Medical Services, a position which he held until 1945 when he went on to the reserve of officers. His valuable qualities and his outstanding achievements were recognized by the award of the O.B.E., a richly merited honour.

RICHARD JENKINS LYONS.

THE University of Sydney is the poorer for the death on November 13, 1951, of Richard Jenkins Lyons, Senior Lecturer in Mathematics and Reader in Geometry. Richard Lyons was not only one of the university's ablest scholars, but one of its most loved and respected teachers. Educated at the Sydney Grammar School, he came up to the University of Sydney in 1903, with the entrance scholarship for mathematics. In 1906 he graduated with first-class honours and the university medal. Two years later he was awarded a travelling scholarship to Cambridge and entered Saint John's College in 1908. He became a wrangler and Fellow of Saint John's College. After his return to Australia, he became lecturer in mathematics and later reader in geometry in the University of Sydney. During the second World War he was appointed to the Department of Naval Intelligence in Melbourne. For this work he learnt Japanese with remarkable speed and efficiency. His superior officer in the service has written not only of his work but of his "incurably merry manner" and of his "strength of spirit".

Throughout a busy life he found time to keep abreast of modern developments in mathematics: in particular with the work of Professor H. F. Baker, of the University of Cambridge. His work was mentioned and acknowledged in Volumes II and III of Baker's "Principles of Geometry". From time to time notes and papers, some in collaboration with others, appeared in the *Mathematical Gazette*, the *Philosophical Magazine* and the *Proceedings of the Cambridge Philosophical Society*. A modest and self-effacing man, he was beloved by all his colleagues, many of whom have spoken and written of the help and inspiration he afforded them in their own work and publications. Richard Lyons was a born teacher and his enthusiasm for his chosen subject never wavered. There was never any trouble about discipline in his classes, however large; somehow the class was always on his side. Generations of students will remember his clear vigorous lectures; they will remember too his merry wit, and his unswerving intellectual honesty, with its corollary, a very acute and active sense of humour.

All these things were the vehicles by which his influence reached a wide and varied circle of friends; of that influence itself, of his essential character it is somewhat more difficult to speak. His whole life was founded and guided by a strong religious faith. He had a quality of detachment, unmistakable but hard to describe. For him the shoddy and cheap things simply did not exist. He was one of the happiest of men, always good-humoured, never uncharitable, though when the occasion called for it he had a certain gift for scorn.

He has left us at an age when he might have—and indeed did—look forward to many years of happy useful retirement. Our sympathy is extended to Mrs. Lyons and to his sister, Dr. Ettie Lyons, of Taralga.

THOMAS HARVEY JOHNSTON.

The following appreciation of the late Professor Thomas Harvey Johnston has been forwarded by Professor J. B. Cleland.

Professor Thomas Harvey Johnston, D.Sc., whose death in his seventieth year occurred suddenly at the home of his son-in-law at Parkside near Adelaide on the morning of August 30, 1951, had held the chair of zoology at the University of Adelaide since 1922. He had consequently over a period of nearly thirty years been responsible for the teaching of the principles of general biology and of parasitology to Adelaide medical students. Those medical graduates who served in the war in tropical areas have been grateful to him for the thoroughness of his teaching of protozoology and helminthology—a thoroughness perhaps hardly appreciated in full at the time. But Professor Johnston's teaching of medical students was not his only close association with our profession. I had myself the good fortune to be associated with him early in his career when he was appointed an assistant microbiologist in the newly created Bureau of Microbiology in Sydney, under the able direction of the late Dr. Frank Tidswell. Here he was responsible for the identification and study of the species of rats and their fleas and other parasites during the later epidemics of plague in New South Wales. He also undertook the description of the parasitic worm, which was called *Spiroptera gibsoni* (after its discoverer, Dr. Gibson, of Windsor), responsible for the worm-nodules so frequent in the briskets of cattle and viewed askance by the meat inspectors of the departments of public health. From his work, commenced at the bureau in 1909, he became a world authority in helminthology, paying particular attention to the cestodes and trematodes and their life-histories, and describing several new genera and a large number of new species of parasitic worms and some protozoa. In South Australia, in addition to his teaching and research work, he was asked by the Minister of Public Works to become a member of the advisory committee on water supplies, which advises the engineering and water supply department on matters affecting the purity of the various public water supplies. For many years a science graduate, under his direction, recorded weekly the algæ and other organisms (apart from bacteria) in the Adelaide water supply—a matter at times, in hot summer periods when algal growth was fostered, of considerable public health importance. His advice was also very welcome in relation to the biological aspects of sewage disposal at the Glenelg treatment works. He was a member of most of the expeditions into the drier parts of South and Central Australia organized by the Board for Anthropological Research of the University, in which he assisted the writer in blood-grouping the natives, took finger and hand prints and samples of hair, and collaborated in the collection of material illustrating the uses made of plants and animals by our aborigines. Apart from these activities with a medical bearing, he accompanied Sir Douglas Mawson on his Antarctic expeditions in 1929-1930 and 1930-1931; he was chairman of the Prickly Pear (Travelling) Scientific Commission of the Queensland Government from 1912 to 1914, and was responsible for the introduction of the cochineal insect that proved so successful in the control of this pest; he was scientific controller of the Commonwealth Prickly Pear Laboratories from 1920 to 1923; he was chairman of the Museum Board of South Australia; he was president and honorary secretary of the Royal Society of South Australia, and was an active member of many other scientific societies.

From my close association with him, first in the Bureau of Microbiology in Sydney and later when he occupied the chair of zoology in Adelaide, I can speak in the highest terms of his sterling qualities as a colleague and as a friend. He was an indefatigable scientific worker as his 250 or more papers show, and this work was remarkably thorough and reliable; he was very orderly in his working laboratory, his study and his home; as a colleague under field conditions, which were sometimes rather strenuous and trying, no one could have helped more or been more considerate; and many little incidents from time to time revealed an ethical code that he lived up to and to which few of us can attain. His long and happy domestic life was saddened by the death of his only son, a member of our profession who had served in the war. His daughter married Dr. W. W. Jolly, of Parkside. An account, more particularly of his scientific work, is being prepared by Sir Douglas Mawson for the Royal Society of South Australia. A bibliography of his contributions to knowledge since 1922 appears in the Calendars of the University of Adelaide.

JAMES MACARTHUR.

WE are indebted to Dr. Maurice Joseph for the following appreciation of the late Dr. James Macarthur.

With the recent death of James Macarthur the profession lost one of its outstanding country practitioners. Born in County Down, Northern Ireland, in 1881, the son of Dr. Peter Macarthur, he qualified with the licentiate of the Royal Colleges of Physicians and of Surgeons of Edinburgh in 1902, and then, after a resident post at the London Hospital, returned to the Royal University of Ireland (now

Queen's University), where he graduated M.B., Ch.B., B.A.O. in 1905. That year he migrated to Australia and practised in the northern coal districts of New South Wales until 1915, when he returned to England to join the Royal Army Medical Corps, in which he served till the armistice. He was gassed in France, and this was the genesis of a chronic respiratory ailment, which troubled him for the rest of his days. In 1919 he returned to Australia and practised at Pambula. During his three years there he studied to such good purpose that in 1922 he visited Edinburgh and gained his Fellowship of the Royal College of Surgeons. Returning once again to his adopted country, he practised at Cessnock and Muswellbrook, and in 1934 he moved to Bombala, where he worked until his death.

Dr. Macarthur was a man of great capabilities, and his reputation, especially as a surgeon, attracted patients from a wide area in the southern districts of New South Wales. Thoroughness was the keynote of all his work, and he spared no pains to keep abreast of modern medical practice. He was essentially an individualist, and I doubt whether any better one-man diagnostic service existed in the whole country. He was equipped with an X-ray plant and an electrocardiograph and efficiently performed the ordinary hæmatological and biochemical examinations, using for the latter mostly modern colorimetric methods. Despite a busy practice, made more arduous by these additional services, he found time to be interested in civic matters and was mayor of Bombala for six years during the war as well as being an active member of the Volunteer Defence Corps. His hobby was wood and metal turning, and from a well-equipped workshop complete with electrically driven lathes he turned out a variety of articles, including a model railway.

His life, which encompassed half a century of devoted medical service, was indeed a full one. During his last four years he had the pleasure and satisfaction of taking his son, Dr. R. J. Macarthur, into his practice. He married in 1911 and is survived by his widow, son and daughter.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the Eighth Session of the Australasian Medical Congress (British Medical Association) to be held at Melbourne from August 22 to 29, 1952, has sent the following information for publication.

Programme of Scientific Meetings.

The following is the programme of scientific meetings to be held on Tuesday, August 26, and Wednesday, August 27, 1952.

Tuesday Morning, August 26.

Hypertension (Panel Session).—Chairman: Professor E. S. J. King (Victoria). The following speakers will take part in the discussion: Dr. F. B. Byrom (New South Wales), Dr. Alex Murphy (Queensland), Dr. I. Douglas Miller (New South Wales), Dr. Kevin O'Day (Victoria), Dr. R. H. Orton (Victoria).

Clinical Problems of the Menopause.—Chairman: Professor Bruce Mayes (New South Wales). Speakers: Dr. J. W. Johnstone (Victoria), "Use and Abuse of Hormones in Therapeusis of the Menopause"; Dr. Ellen Kent Hughes (New South Wales), "Management of Menopausal Patients in the Home"; Dr. C. Chapman (New South Wales), "Problems Associated with Artificial Menopause"; Dr. G. B. Murphy (Queensland), "Psychology of the Menopause". It is also hoped to arrange for an overseas speaker to take part in this discussion.

A Review of Poliomyelitis.—Chairman: Dr. D. M. McWhae (Western Australia). Speakers: Sir Macfarlane Burnet (Victoria), "Laboratory Aspects"; Dr. M. L. Powell (Victoria), "Diagnosis and Medical Management"; Dr. E. E. Price (Victoria), "Orthopædic Management"; Dr. R. V.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 23, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.[2]	Northern Territory.	Australian Capital Territory.	Australia.[3]
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	2	..	2	..	..	..	..	..	4
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	4	..	..	..	..	1	..	5
Diphtheria	3(1)	4(4)	1(1)	..	3(3)	..	..	..	11
Dysentery (Bacillary)	..	13(13)	1	1(1)	1	..	..	..	16
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1(1)	..	..	..	..	..	..	1
Infective Hepatitis	..	..	..	..	7(5)	..	..	..	7
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	3	..	..	..	3
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1(1)	2(1)	..	..	3	..	..	..	6
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	8(4)	2	6(2)	23(14)	..	..	..	..	39
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	3(3)	..	..	..	..	..	..	3
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	20(10)	23(13)	2	..	..	..	..	1	46
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	45(31)	11(8)	11(8)	14(13)	8(7)	..	..	..	89
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	1(1)	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.
[2] Figures not available.
[3] Figures incomplete owing to absence of returns from Tasmania.

Southcott (South Australia), "Epidemiological Precautions"; Dr. Douglas Galbraith (Victoria), "Rehabilitation".

Tuesday Afternoon, August 26.

Uses of Cortisone and ACTH.—Chairman: This appointment has not yet been made. Speakers: Dr. K. S. Harrison (New South Wales), "Use of Cortisone in General Medical Disease"; Dr. R. Strang (Victoria), "Indications and Results in Arthritis"; Dr. E. V. Barling (New South Wales), "Indications of Cortisone and ACTH in Surgery". It is hoped that there will be an overseas speaker to take part in this discussion as well.

Recent Advances in Infectious Diseases.—Chairman: Dr. H. McLorinan (Victoria). Speakers: Dr. A. Fryberg (Queensland), "Epidemiology"; Dr. N. J. Symington (New South Wales), "Therapeutics"; Dr. Leigh Cook (Western Australia), "Isolation and School Problems". It is hoped to arrange for another speaker to discuss the bacteriological aspects.

Wednesday Morning, August 27.

Mass Management of Burns.—Chairman: Sir Samuel Burston (Victoria). Speakers: Dr. R. J. Walsh (New South Wales), "Resuscitation as a National Problem"; Brigadier A. W. Wardell (Victoria), "The Problems to be Expected from the Point of View of Civil Defence"; Colonel Refshauge (Victoria), "Organization of the Medical Services"; Dr. J. W. Perry (Victoria), "The Organization and Use of Laboratory Technique". It is hoped that another speaker will discuss the surgical aspects.

The Medical Care of the Aged.—Chairman: Dr. W. W. S. Johnston (Victoria). Speakers: Dr. A. B. McCutcheon (Victoria), "Geriatrics as a Social Problem"; Dr. Ralph Whishaw (Tasmania), "Acute Medical Conditions in the Aged"; Dr. J. W. S. Laidley (New South Wales), "Limitations of Surgery in the Aged"; Dr. Gilbert Troup (Western Australia), "Anæsthetic Management of Patients over the Age of Sixty-Five"; Dr. A. T. Edwards (New South Wales), "Psychology of the Aged".

Diagnosis and Treatment of the Chronic Cough.—Chairman: Dr. W. Cotter Harvey (New South Wales). Speakers: Dr. C. Fitts (Victoria), "Diagnostic Possibilities"; Dr. H. D. Sutherland (South Australia), "Investigation of the Chronic Cough"; Dr. A. B. Watkins (New South Wales), "Chronic Cough in the Field of Oto-Rhino-Laryngology"; Dr. R. M. deLambert (New South Wales), "Diagnostic Radiological Technique"; Dr. K. N. Morris (Victoria), "Indications for Physiotherapy".

Wednesday Afternoon, August 27.

A Review of the Antibiotics (Panel Session).—Chairman: Dr. Edgar Thomson (New South Wales). The following speakers will take part in the discussion: Dr. P. L. Bazeley (Victoria), Dr. S. Williams (Victoria), Dr. Alan E. Lee (Queensland), Dr. R. B. Charlton (Queensland), Miss Jean Tolhurst (Victoria).

Pruritus: General and Local.—Chairman: Professor W. S. Dawson (New South Wales). Speakers: Dr. I. Stahle (Victoria), "Diagnosis"; Dr. A. J. M. Sinclair (Victoria), "Psychosomatic Problems"; Dr. Douglas Gordon (Queensland), "Industrial Medicine"; Dr. J. J. Searby (Victoria), "General Practice Problems". It is hoped to have a dermatologist to discuss the aspect of treatment.

Australian Medical Board Proceedings.

TASMANIA.

THE following have been registered, pursuant to the provisions of the *Medical Act*, 1918, as duly qualified medical practitioners: Cornish, Geoffrey James, M.B., B.S., 1951 (Univ. Melbourne); Green, Ruth Helen, M.B., B.S., 1951 (Univ. Sydney); MacFie, Hugh Blyth Alexander, M.B., Ch.B., 1936 (Univ. Edinburgh), D.T.M. and H., 1940 (Univ. Edinburgh); Andrea, Bryan Maxwell, M.B., B.S., 1951 (Univ. Sydney); Lewis, John Malcolm, M.B., B.S., 1952 (Univ. Sydney); Duggan, John Malcolm, M.B., B.S., 1952 (Univ. Sydney).

Medical Appointments.

Dr. D. O. Crompton has been appointed honorary assistant ophthalmologist at the Royal Adelaide Hospital in the Hospitals Department.

Dr. W. H. Baudinet and Dr. J. S. T. T. Hill have been appointed honorary medical officers at the Mount Gambier Hospital, South Australia.

Dr. J. S. Flett has been appointed honorary medical officer at the Wallaroo Hospital, South Australia.

Dr. John Richard Carter has been appointed quarantine officer, Northern Territory, under the provisions of the *Quarantine Act*, 1908-1950.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cotton, Brian Drummond, M.B., B.S., 1950 (Univ. Sydney), 6 Lisgar Road, Hornsby.

Summerbell, Edward Britton, M.B., B.S., 1950 (Univ. Sydney), 125 Darling Point Road, Darling Point.

Burcher, Noel Frederick, M.B., 1950 (Univ. Sydney), Captain's Flat, New South Wales.

Diary for the Month.

MARCH 25.—New South Wales Branch, B.M.A.: Council Quarterly.

MARCH 26.—Victorian Branch, B.M.A.: Council Meeting.

MARCH 27.—New South Wales Branch, B.M.A.: Annual Meeting.

MARCH 27.—South Australian Branch, B.M.A.: Scientific Meeting.

MARCH 28.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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